

THE MEDICAL JOURNAL OF AUSTRALIA

VOL. I.—32ND YEAR.

SYDNEY, SATURDAY, MARCH 31, 1945.

No. 13.

Table of Contents.

[The Whole of the Literary Matter in THE MEDICAL JOURNAL OF AUSTRALIA is Copyright.]

ORIGINAL ARTICLES—	Page.	BRITISH MEDICAL ASSOCIATION NEWS—	Page.
Rubella during Pregnancy of the Mother, with its Sequelæ of Congenital Defects in the Child, by N. McAlister Gregg	313	Scientific	332
Congenital Deaf-Mutism as a Sequela of a Rubella-Like Maternal Infection during Pregnancy, by D. G. Carruthers	315	Notice	334
The Influence of Malaria on the Kline and Complement Fixation (Wassermann) Tests for Syphilis, by C. B. Cox and M. J. Durant	320	POST-GRADUATE WORK—	
REPORTS OF CASES—		Courses at Melbourne during 1945 for Medical Graduates	334
Aneurysm of the Ventricle of the Heart, by T. J. F. Frank	325	CORRESPONDENCE—	
REVIEWS—		"Found Dead", "Dead in Bed", and "Collapsed and Died"	334
A Manual for Patients with High Blood Pressure	326	Injuries by Unknown Agents to Bathers in North Queensland	334
A Year Book on Medicine	326	The Treatment of Hemorrhoids by Injection	335
LEADING ARTICLES—		A Disclaimer	335
Ophthalmological Research: Hopes for the Future and a Task for the Present	327	Bell's Palsy	335
CURRENT COMMENT—		NAVAL, MILITARY AND AIR FORCE—	
Measles	328	Appointments	335
Blood Changes in Primary Atypical Pneumonia	329	Casualties	336
Biopsy of the Thyroid	329	OBITUARY—	
ABSTRACTS FROM MEDICAL LITERATURE—		Samuel Joseph Henry Moreau	336
Physiology	330	Cedric James Logan	336
Biochemistry	331	NOMINATIONS AND ELECTIONS	336
		MEDICAL APPOINTMENTS	336
		BOOKS RECEIVED	336
		MEDICAL APPOINTMENTS: IMPORTANT NOTICE	336
		EDITORIAL NOTICES	336

RUBELLA DURING PREGNANCY OF THE MOTHER, WITH ITS SEQUELÆ OF CONGENITAL DEFECTS IN THE CHILD.¹

By N. McALISTER GREGG,
Sydney.

THE ophthalmological aspects of the subject chosen for this joint meeting of the Section of Pædiatrics and the Section of Oto-Rhino-Laryngology of the New South Wales Branch of the British Medical Association have been discussed in some detail in two previous papers.⁽¹⁾⁽²⁾ I propose, therefore, to deal with the subject on this occasion on rather general lines. It may be of interest briefly to recall the manner in which the presence of congenital defects in the infant was first linked up with an attack of rubella in the mother during pregnancy.

In the early months of 1941, cases of an unusual type of congenital cataract made their appearance in Sydney. Although one was struck with the novel character of these cataracts in the first few cases, it was only when other similar cases continued to appear that serious thought was given to their causation. The remarkable similarity of the opacities in the lens, the frequency of an accompanying affection of the heart and the widespread geographical incidence of the cases suggested that there was some common factor in the production of the diseased condition, and that the latter was the result of some constitutional disturbance of toxic or infective nature rather than of a purely developmental defect.

The question arose whether this factor could have been some disease or infection occurring in the mother during pregnancy, which had then interfered with the developing cells of the lens. By a calculation from the date of the birth of the baby it was estimated that the early period of pregnancy corresponded with the period of maximum

intensity of the widespread and severe epidemic of German measles in 1940. Special attention was accordingly paid to the history of the health of the mothers during pregnancy, and in each new case it was found that the mother had suffered from that disease early in her pregnancy, most frequently in the first or second month. In some cases she had not at that time yet realized that she was pregnant. The investigation was then repeated in the early cases in which such a history had not been sought, and again the history of early German measles infection was obtained. Moreover, in all these cases the health of the mother during the remainder of the pregnancy was described as good. As the constant involvement of the central nuclear fibres in the cataractous process suggested an early incidence of the noxious factor, it was considered that a possible solution of the problem had been obtained. Confirmation of this theory was therefore sought from any of my colleagues who had seen lesions of this type, and they kindly agreed to assist me by inquiry into the health of the mothers during pregnancy. The result of their inquiries confirmed the amazing frequency of the German measles infection.

The matter was then put before the Ophthalmological Society of Australia (British Medical Association), and a questionnaire was sent out to members. From the reports furnished in response to this questionnaire, together with reports of cases supplied by the sisters in charge of baby health centres in New South Wales, and from my own series of thirteen cases, a total of 78 cases was collected. This furnished the material for the paper "Congenital Cataract following German Measles in the Mother",⁽³⁾ which I read at the third annual general meeting of the Ophthalmological Society of Australia (British Medical Association) in October, 1941.

The special ocular features noted in this group of cases were the following: (a) cataract, usually bilateral and of an unusual type which I called subtotal; (b) nystagmus, or rather coarse, jerky, purposeless movements of the eye-balls; (c) microphthalmia; (d) weakness and sluggishness of the pupillary reaction to light, and in some cases a

¹ Read at a meeting of the New South Wales Branch of the British Medical Association on December 14, 1944.

somewhat atrophic appearance of the irides; difficulty of obtaining and maintaining mydriasis. Vision, of course, was defective, but the babies appeared to follow readily any movement of the light source.

In addition to these ocular manifestations there were some other striking features. Most of the babies were of small size, ill-nourished and difficult to feed, with the result that many of them came under the care of the paediatrician before being seen by the ophthalmic surgeon. Many of them were found to be suffering from a congenital defect of the heart. In addition, intolerance to atropine was present in many of the cases; rises of temperature up to 105° F. were not uncommon after the instillation of atropine drops. This extreme reaction to atropine was probably due to the dehydrating effect of the drug on the already emaciated, ill-nourished infant.

At the time of reading of the paper 15 of the 78 patients had died, and since that date several more deaths have occurred. Many of the patients showed mental as well as physical retardation. As has previously been mentioned, an extremely high percentage of these babies had a congenital defect of the heart; in my own series of cases it was present in all but one.

Dr. Margaret Harper kindly supplied me with the following description of eight patients examined by her:

All these babies were seen because of difficulty in feeding and failure to thrive. They all had symptoms suggesting a cardiac defect such as difficulty in taking the breast; they had to be fed in their cots by bottle and some by gavage. They were all in the acyanotic or potentially cyanotic groups of cardiac defects. None was cyanotic. There was a harsh systolic murmur over the base of the heart and down the sternum in all. Some had a thrill. All had signs suggesting the continuance of a fetal condition or of a malformation of the heart.

Autopsy, carried out in three cases at the Royal Alexandra Hospital for Children, revealed a wide patency of the *ductus arteriosus*. In other cases septal defects have been noted. In a few cases in which the heart was clinically reported as unaffected, post-mortem examination revealed a patent *ductus arteriosus*.

A further complication noted in a few of my cases was the development of a dry, scaly, eczematous condition involving the face, scalp and limbs, which was very resistant to treatment.

Although the ocular and cardiac defects were the most striking features in these early cases, it was thought, and the opinion was expressed, that other defects were likely to appear as time passed. The first new complication which came to my notice was deafness. On December 6, 1941, there appeared in THE MEDICAL JOURNAL OF AUSTRALIA a leading article on "Congenital Cataract following Rubella", in the course of which my paper was reviewed. The local Press in turn extracted this leading article, and during the following week I had telephone calls from three mothers, informing me that they had suffered from German measles early in pregnancy; each thought that her child was deaf, and each wished to know if this deafness could have resulted from the infection. Dr. Eric Blashki kindly saw these patients for me, and he said that, although they were too young for any proper testing, so far as he could say they appeared to be deaf.

In the course of the succeeding two years, 1942 and 1943, more and more of these cases of "deaf-mutism" appeared. Their appearance at a later stage than cases with ocular complications is natural, because deafness would not be suspected even by the mothers until a much later date than blindness. During this same period many babies were brought to the paediatrician because of failure to thrive, restlessness and signs of backwardness.

The next development in the investigation of this question took place in South Australia. The Institute of Medical and Veterinary Science, Adelaide, sponsored a committee under the able leadership of Dr. Charles Swan. The committee had the following objectives:

... to confirm and if possible to extend Gregg's findings, and to determine: (i) whether the disease during pregnancy was rubella or some illness which simulated it; (ii) what was the precise period of preg-

nancy in which the disease was effective in producing congenital abnormalities; (iii) what was the full range of the defects; and (iv) whether other infectious diseases occurring during pregnancy led to similar effects.

The committee's findings have been presented in two excellent contributions⁽¹³⁾⁽¹⁴⁾ appearing in THE MEDICAL JOURNAL OF AUSTRALIA on September 11, 1943, and on May 6, 1944. The members of the committee confirmed the original findings and drew attention to other sequelae—deaf-mutism with or without cardiac disease, cardiac disease without any other defect, and microcephaly. Two of their conclusions are worthy of mention. In one they state that "clinically we have little doubt that the exanthematous disease which occurred in our cases was 'German measles'", and in the second, "we have obtained no evidence of any condition other than rubella as an antecedent of the congenital defects we describe".

In THE MEDICAL JOURNAL OF AUSTRALIA of September 2, 1944,⁽¹⁵⁾ Dr. Mervyn Evans, from the same institute, published a paper describing some dental defects noted in some of these cases. In the *American Journal of Ophthalmology*, May, 1944,⁽¹⁶⁾ Dr. A. B. Reese, of New York, wrote an article in which he described three cases of congenital cataract accompanied by congenital cardiac defect, in which the mothers had suffered from rubella early in pregnancy. He described the cases as similar to those in the Australian reports. The epidemic occurred about the end of 1942. Two cases were reported in *The Lancet* of April 8, 1944,⁽¹⁷⁾ and I have received several communications from the United States of America from doctors and also from parents describing cases, some of ocular defects, others of deaf-mutism, similar in all respects to those of our own series, and all accompanied by cardiac defect. In every case there was the history of rubella in the first or second month of pregnancy. In *The Journal of Pediatrics*, October, 1944,⁽¹⁸⁾ C. A. Erickson contributed an article entitled "Rubella Early in Pregnancy Causing Congenital Malformations of Eyes and Heart".

Then, in 1943, I had a further series of eight cases. The mothers contracted rubella during the 1942 epidemic of that exanthem. The appearance of this second series of cases following another epidemic after an interval of two years is, I think, of great significance. In this series the ocular defects noted corresponded closely with those encountered in the previous series. There were six cases of cataract and one of hydrophthalmia, whilst in the eighth case unilateral microphthalmia was present. Cardiac disease was present in six of the eight cases. In three cases deaf-mutism was present as an additional complication. In this series two deaths have occurred.

I have recently had the opportunity of examining several children afflicted by deaf-mutism without any apparent ocular defect. The parents have in all cases stated that the child appeared to have good vision. In many of these cases striking changes have been detected in the fundi; these are of the nature of well-marked pigmentary disturbances most frequently in the central region. In other cases other areas of the fundi were also involved. It is proposed to examine these cases in more detail and to record the findings.

What is the causal mechanism of production of the congenital defects? This is essentially a question for the embryologist. At the annual meeting of the Ophthalmological Society of Australia (British Medical Association), held at Melbourne in October, 1944, one paper read was entitled "Some Embryological Observations on Congenital Cataract Associated with Rubella in the Mother".⁽¹⁹⁾ This was contributed by Miss Ida Mann, the Margaret Ogilvie Reader in Ophthalmology at Oxford. Miss Mann expressed the opinion that it had been proved beyond question that there was a causative relationship between a virus infection early in pregnancy and the appearance of certain congenital defects in the infant. She said that in man the most important determining factor for the effect of a maternally transmitted disease was the time of its action, only those cells which were in active division being affected. She then proceeded to consider the times of active cell division in the lens, the internal ear and the heart, and the relation of these times to the times of illness of

the mothers of affected children. With regard to the lens, Miss Mann found that the type of opacity and the time of the mother's illness were complementary. In the cases of partial deafness she considered that the time of the illness in the mother and its connexion with the defect produced was borne out also. The cardiac anomalies, she considered, were of the nature of arrests, and were quite compatible with a showing of differentiation at the appropriate time.

Another development I should like to record is that at the request of the Director-General of the Department of Public Health of New South Wales, a committee has been formed to investigate all aspects of congenital defects following maternal rubella. This committee is composed of Dr. E. S. A. Meyers, representing the Department of Public Health, Dr. Donald Vickery, Dr. Ramsay Beavis, Dr. Mary Heseltine and myself. A questionnaire was printed in the monthly notices sent out by the New South Wales Branch of the British Medical Association, and preliminary reports have been collected of 129 cases of congenital defects.

Another committee was convened by Dr. A. E. Machin, Principal Medical Officer of the Department of Public Instruction of New South Wales, to consider the training and subsequent education of these children. The first problem was to divide the children into two groups—(a) the educable, (b) the ineducable. As a first step in this direction, a full survey of as many as possible of the children is being carried out by representatives of both these committees. The Board of the Royal Alexandra Hospital for Children, through the general superintendent and chief executive officer, Dr. S. W. G. Ratcliff, has kindly placed the out-patient department at our disposal, and the survey is being carried out at special sessions on Saturday mornings. We are indebted to the hospital also for arranging for radiographic examination of the heart in all cases. The results of the survey will, I imagine, be published in due course.

Without in any way forestalling such a publication, as chairman of the committee, I may state that we feel that the routine radiological examination of the heart has been justified. The committee has been particularly gratified by the extreme interest and keenness shown by parents, who are particularly anxious to assist in every possible way; we are receiving requests from the parents of patients hitherto unreported, to be allowed to take their children to be examined.

It is difficult to forecast the future for these children. So many of them have retardation of their physical and mental development that the outlook is doubtful. I have noticed in some cases an undoubted improvement in both physical and mental standard in the past six months; if this progress continues, then some patients previously regarded as unfit for training will become suitable for training and tuition at least in certain avenues. Broadly, it would appear that the outlook is better for the patients with deaf-mutism than for those with ocular defects. The question of operation in cases of patent *ductus arteriosus* must also be considered in this regard.

Conclusion.

Though some progress has been achieved, I think that much still remains to be done, not only in respect of rubella and its sequelæ, but also with regard to congenital defects in general. In regard to rubella, it would appear important (i) to isolate the virus and, if possible, to develop treatment both prophylactic and curative; (ii) to arrange for more publicity amongst the medical profession and the public, with the object of preventing infection of the potential mother. With regard to congenital defects in general, may not diseases other than rubella have a harmful effect on the developing embryo? May it not be possible that a thorough, careful and detailed investigation of the maternal history throughout pregnancy will help us to unravel some of our unsolved problems? Mongolism seems to me to be one disease in which research on these lines may be worth while. In conclusion, I regard "rubella and its sequelæ" as the title of but one chapter in the full story of congenital defects; many more chapters have yet to be written.

References.

- (1) N. McA. Gregg: "Congenital Cataract following German Measles in the Mother", *Transactions of the Ophthalmological Society of Australia (British Medical Association)*, Volume III, 1941, page 35.
- (2) N. McA. Gregg: "Further Observations on Congenital Defects in Infants following Maternal Rubella", *Transactions of the Ophthalmological Society of Australia (British Medical Association)*, Volume IV, 1944, in the press.
- (3) C. Swan, A. L. Tostevin, the late B. Moore, H. Mayo and G. H. Barham Black: "Congenital Defects in Infants following Infectious Diseases during Pregnancy", *THE MEDICAL JOURNAL OF AUSTRALIA*, Volume II, 1943, page 201.
- (4) C. Swan, A. L. Tostevin, H. Mayo and G. H. Barham Black: "Further Observations on Congenital Defects in Infants following Infectious Diseases during Pregnancy, with special Reference to Rubella", *THE MEDICAL JOURNAL OF AUSTRALIA*, Volume I, 1944, page 409.
- (5) M. W. Evans: "Congenital Dental Defects in Infants Subsequent to Maternal Rubella during Pregnancy", *THE MEDICAL JOURNAL OF AUSTRALIA*, Volume II, 1944, page 225.
- (6) A. B. Reese: "Congenital Cataract following German Measles in the Mother", *American Journal of Ophthalmology*, Volume XXVII, May, 1944, page 483.
- (7) R. E. Hope Simpson: *The Lancet*, April 8, 1944, page 483.
- (8) C. A. Erickson: "Rubella Early in Pregnancy Causing Congenital Malformations of Eyes and Heart", *The Journal of Pediatrics*, October, 1944, page 281.
- (9) I. Mann: "Some Embryological Observations on Congenital Cataract Associated with Rubella in the Mother", *Transactions of the Ophthalmological Society of Australia (British Medical Association)*, Volume IV, 1944, in the press.

CONGENITAL DEAF-MUTISM AS A SEQUELA OF A RUBELLA-LIKE MATERNAL INFECTION DURING PREGNANCY.

By D. G. CARRUTHERS,
Sydney.

An exanthem, believed to be rubella, was experienced in widely scattered areas of Australia, especially during the years 1940, 1941 and 1942. Case reports indicate that the disease was in rising frequency in 1939, and sporadic cases occurred even in 1937, 1938 and again in 1943. Just what has happened since, the public health authorities may later inform us. A peculiarity of the epidemic stage of this rubella was a greater tendency than usual for adults, and especially young adults, to become infected. A more dire result of this has been that a proportion of pregnant women contracted the disease, which, though of minor importance so far as the mother was concerned, has been disastrous in its effects upon the developing fetus, as revealed in the infant born later. Congenital cataracts, patent *ductus arteriosus* and other cardio-vascular defects, deaf-mutism, microcephaly, and general stunting of growth and dental defects have been recorded.

Gregg,⁽¹⁾ in October, 1941, recorded a series of 78 cases of congenital cataract occurring in babies. In almost all of these cases the mother had suffered from rubella during the early months of pregnancy. Gregg also recorded the fact that many of these babies were of small size and were difficult to feed. A congenital heart lesion was also noted in a large number of this series. I do not think that Gregg at this stage mentioned deaf-mutism.

The Incidence of Deaf-Mutism.

In September, 1943, from South Australia, Swan and others⁽²⁾ reported on a group of 49 cases of rubella occurring during pregnancy. Thirty-one of these women had subsequently given birth to children with various developmental defects. These 31 mothers had, with but two exceptions, contracted rubella within the first three months of pregnancy. Of the 31 children, deaf-mutism was recorded in seven. In a further report published in May, 1944, Swan and his colleagues recorded an additional twelve cases of rubella in pregnancy. In ten of these the infection occurred within the first three months, and in all ten the offspring exhibited abnormalities. In two other instances the mothers had been infected during the fifth and sixth months respectively, and their babies were completely

¹ Read at a meeting of the New South Wales Branch of the British Medical Association on December 14, 1944.

normal. Of this later group of ten abnormal children, no fewer than five are deaf mutes. Research into the incidence of deafness is seen in truer perspective when it is revealed that the seven deaf-mute children of the first series were all aged two and a half years or older when the records were made. In this age group there were also seven children who were not deaf mutes, so that of the age group one-half were deaf and dumb. In the later group of ten afflicted children, the five who were recorded as deaf mutes were all then over three years of age. It is necessary to realize that deafness may often be overlooked in the very young baby, especially if there is something else obviously and seriously amiss, such as opaque lenses, general bodily stunting or difficulty in maintaining nutrition. Unless deafness is deliberately sought, it is often not until speech development is obviously retarded that the anxious parents seek advice, and then the true situation, the presence of deafness and secondary mutism, is revealed. We may anticipate, I think, that a later survey of the earlier published lists will reveal more children who are deaf. It is possible, too, that still later in life audiometric tests may reveal partial deafness in many. One cannot state yet exactly what proportion of these children, born of mothers who have suffered from rubella during pregnancy, have severe defects of hearing, although it is likely that the figure is over 50%.

It is my privilege to present on behalf of my colleagues in otology an analysis and some comment upon relevant details which have been observed in the study of deaf-mute children whose mothers were infected with rubella during pregnancy. An attempt has been made to collect all the cases together, with the help of the enthusiastic efforts of Dr. E. S. A. Meyers, of the New South Wales Department of Public Health. Dr. Meyers informs me that as a result of the departmental appeal to doctors, 147 cases of congenital defects have been reported; of this series, a history of maternal rubella was recorded in 102. Twenty-two mothers denied any such infection, and in 23 cases the history is not yet certain. Of the 147 children, severe deafness is present in 116. Of the 102 whose mothers are known to have suffered from rubella, 74 are deaf, and 14 of these also have congenital heart lesions. The percentage of congenital deafness is thus even higher in the Board of Health figures than in earlier reports. It has not been possible for me to tabulate the details of the whole series of 116 deaf children reported to the Board of Health. Seventeen patients have been under my private care, however, and I feel that from these I can present a summary which will be applicable to the whole series. In addition, I have had under my care five deaf-mute children of similar age group, but in regard to whom the maternal infection is not evident. Although I suspect that the deaf-mutism of these five also follows maternal rubella, I have separated them in the analysis and I have omitted them from the series in attempting to come to conclusions.

Relation of the Period of Pregnancy at which Rubella Occurred and the Intensity of the Illness, to the form of Abnormality to be Found in the Child.

Analyses have been made to determine whether the stage of pregnancy at the time of infection of the mother, or the intensity and duration of the mother's illness, bears any relation to the nature and degree of congenital abnormalities subsequently to be found in the child. Swan and his colleagues report that in the deaf-mute children of their series the average period of pregnancy when maternal infection occurred was 2.3 months, whereas the average duration of pregnancy of the mothers whose children suffered from cataract was 1.5 months. These writers stressed the evidence that the type of congenital defect is dependent upon the stage of pregnancy at which the mother suffers from rubella. In my own series of deaf-mute patients, if we except Case II, in which infection was said to have occurred during the sixth month, the average stage of pregnancy works out at about the second month. While this is in keeping with the belief that the term of pregnancy at the time of infection is of some significance in deciding the nature of developmental defects, nevertheless, it must be recognized that the taking of an average

may be misleading, when, in a process so regular to timetable as foetal development, one finds evidence of maternal infection at one month, at three and a half months and apparently at six months, in the case of deaf-mute offspring. I am inclined to think that the importance of the stage of pregnancy at which infection occurs is that, if it is in the first six weeks, foetal damage will be widespread and may include the eyes, both divisions of the ears, the heart and perhaps many other parts. After the sixth week the eyes may escape, the heart may be spared and the semi-circular canals may become normally developed; but the cochlea is still likely to be damaged, and growth may be retarded. After the third month damage to the foetus is rare.

As to the influence of intensity and duration of the maternal infection, an analysis of the details gives surprising results; some of the most intensely affected mothers were ill for periods of seven to fourteen days, yet their infants (Cases III, IV, VI and VII) are no more severely afflicted than are those from mothers who were only mildly ill for as little as one day (Cases I, V, XI, XIII and XIV). Surprising as this observation is, I think it may be of significance in investigations of the problem of how the virus or toxin acts to inflict such damage on the developing foetus. I shall leave that aspect to some other occasion, when perhaps one's theories may be able to be supported by the studies of the laboratory worker. I think, too, that this fact, that a mild and short maternal infection may inflict foetal damage as extensive as that from a severe and prolonged attack, is strongly in support of my impression that many of the other cases of congenital defects, which are otherwise so similar, but lack a history of maternal illness, are due to the same virus.

The Nature of the Deafness and Mutism.

The children of this series are mute because they do not hear. There is no question of aphasia as a reason for failure to speak in any of the children whom I have tested. Although often stunted and frequently microcephalic, these children are all bright and display keen interest in everything; they speedily learn to use such items as an electric torch, a pencil, a tuning fork *et cetera*, and often show early interest in lip-reading.

Classification of the Type of Deaf-Mutism and a Comparison with the Forms of Grouping Determined by Survey of Deaf-Mute Children.

It has been my task to endeavour to classify the type of deaf-mutism which this rubella-like maternal disease has induced. The accepted modes of grouping have been based largely upon aetiological grounds, although these factors have been far from precise, especially in congenital cases. Further classification has also been attempted, based upon clinical study of the particular case, and occasionally after post-mortem study. A child who reaches an age at which congenital deaf-mutism can be definitely recognized, does not perish of the disease. Kerr Love⁽¹⁾ wrote in 1921: "The thing most wanted from the pathologist at present is a series of post-mortem examinations of undoubtedly deaf-born children." This is still true in 1944. Post-mortem records there have been, but most of these have been of children with deafness acquired after birth, or of congenital deaf-mutes, many of whom had survived well into middle age before the opportunity appeared to examine serial sections of the auditory apparatus.

A search of the literature reveals that the late J. S. Fraser, in 1922,⁽²⁾ made the most recent dissection of types and a comparison of various earlier efforts to classify deaf mutes. It is evident that there was then, and there still is, some room for controversy as to the most logical mode of classification.

In an effort to see whether the present series of cases will fit in with the previously recognized types of deaf-mutism, it is necessary briefly to outline the mode of classification. Some are born deaf—congenital deaf mutes; others become deaf in infancy. In both the "congenital" and the "acquired" groups it is possible to recognize two major aetiological forms. In one the tendency to become deaf is associated with constitutional factors, which may

TABLE I.

Case Number and Initials.	Length of Time Pregnant at Onset of Disease.	Date of Maternal Infection.	Intensity and Duration of Maternal Rubella.	Term of Birth. (Months.)	Weight of Baby at Birth.	Congenital Defects.	Progress.	Caloric Labyrinthine Reactions.
1. M.G.	2-3 months.	Oct., 1938.	Slight, 3 or 4 days.	9	lb. oz. 5 4	Deaf mutism, undersized.	Remains totally deaf, microcephalic and stunted at 4½ years.	Reduced response. Right and left. Not disturbed at all.
2. B.B.	6 months.	Oct., 1940.	Slight, 1 week.	9	7 9	Deaf mutism, undersized.	Hears very little, cannot assess tonal capacity. At 3½ years weighs 30 pounds.	Reduced response. Not disturbed at all.
3. J.P.	3½ months.	Oct., 1940.	Severe coryza, 7 days.	9	7 5	Deaf mutism.	At 3 years speaks many words. Seems to hear very little. Normal growth.	Not tested.
4. E.H.	1½ months.	Aug., 1940.	Moderately severe, 10-14 days.	9	6 4	Deaf mutism.	Hears very little. Lips many words. Normal growth at 3½ years.	Normal activity. Right and left. Not disturbed.
5. J.D.	2 months.	Sept., 1940.	Slight, 1 day.	7	4 3	Deaf mutism, microcephalic, stunted.	Hears very little. Growth retarded at 3½ years.	Active right less than left.
6. S.C.	7 weeks.	Oct., 1940.	Moderately severe, 7-10 days.	9	9 5	Deaf mutism.	Normal growth. Hears 512, 1024, 2048 slightly; aged 3 years.	Active, reduced. Not disturbed.
7. C.G.	3 months.	Nov., 1940.	Moderately severe, 7-10 days.	8½	4 10	Deaf mutism.	Normal growth. Hears very little, but attentive to audiometer, especially left ear. Aged 3½ years.	Right reduced, left normal.
8. N.C.	5-6 weeks.	Aug., 1940.	Slight, 1 week.	9	4 14½	Deaf mutism.	Growth retarded. Hears very little. Uses voice, but no words. Aged 3½ years.	Not tested.
9. T.S.	3 months.	Sept., 1941.	Moderately severe, 10 days.	9	5 10	Deaf mutism, stunted.	Growth retarded. Hears little to nothing. Bright and quick to learn at 3½ years.	Not tested.
10. J.L.	2½ months.	Oct., 1940.	Moderately severe, 8 days.	8½	4 12	Deaf mutism, stunted.	May hear 512 in left ear. Intelligent, tries to lip words; aged 3 years.	Normal.
11. W.B.	1 month.	Aug., 1940.	Slight, 2-3 days.	9	3 11	Deaf mutism, stunted.	Growth retarded. May hear 512, 1024, 2048; aged 3½ years.	Not possible.
12. M.T.	5 weeks.	June, 1940.	Slight.	8	4 12	Deaf mutism, stunted.	Hears quite a lot, throughout tone range. Speaks many words. Undersized. Strabismus corrected with glasses. Aged 3½ years.	Not tested.
13. L.G.	6 weeks.	1940.	Slight, 2-3 days.	9	6 8	Deaf mutism, microcephalic, stunted.	Hears very little. Uses voice, but produces very few words. Undersized. Aged 3½ years.	Not tested.
14. L.W.	2 months.	Nov., 1940.	Slight, 1 day.	8	6 8	Deaf mutism, slightly undersized.	Slightly attentive to 512, 1024, 2048, especially right ear. Speaks a few words; aged 3 years.	Reduced right and left, no disturbance.
15. M.J.	2 months.	Sept., 1940.	Severe, 1 week.	8½	6 5	Deaf mutism, stunted, microcephalic.	Hears 512, 1024, 2048 right and left. Speaks a few words, being taught partly by lip-reading; aged 3½ years.	Not tested.
16. M.B.	2 months.	Nov., 1937.	Slight.	9	5 0	Deaf mutism, stunted.	Hears very little, says a few words. Remains stunted at 6 years.	Not tested.
17. G.S.	6 weeks.	July, 1940.	Moderately severe, 1 week.	9½	7 12	Deaf mutism.	Hears 512, 1024, 2048, 4096 right and left very slightly. Uses voice but says no words. Aged 3½ years.	Left slight deviation of eye to left only. Right slight fine nystagmus and deviation to right; no upset.
18. A.C.*	1 month.	August, 1937.	Moderately severe.	8	5 4	Deaf mutism, undersized, congenital heart defects.	Hears quite a lot. Talks indistinctly.	Not tested.

* The assessment of hearing capacity under the heading "progress" was made in Cases 3 and 13 about six months before preparation of this report for printing. Both these children were examined again after the manuscript was prepared and when they were aged nearly four years. In both cases hearing tests now reveal attention to and interest in audiometer notes between 512 and 4,096 cycles. Deafness is still obvious, however. The improvement is probably due to the fact that these children, who are both receiving special teaching, have been made interested in the act of listening and so make use of vestigial receptive power. It seems unlikely that a late development of the inner ear may take place.

* Since this paper was read, the child A.C. (Case 18) has been examined. This case is interesting as another instance in which the maternal infection occurred in 1937.

be inherited. The other includes cases of deaf-mutism due to extraneous influences such as infection, toxins and trauma. It is clear that we can fit these post-maternal rubella cases into the "congenital" group.

Fraser, in his report in 1922, included an analysis of the histories of 135 deaf mutes at the Edinburgh Royal Institution for Education of the Deaf and Dumb. He was able to divide the children into 75 congenital deaf mutes and 60 with deaf-mutism acquired in early life. In only 24 of the congenital cases was there any familial history of deafness or any other possible hereditary factor, and this was by no means of undoubted significance in the particular cases. That left at least 51 cases in which some extraneous cause apparently was responsible. These figures agree substantially with the averaged figures of numerous other

investigators, as quoted by Fraser; that is to say, that slightly over 50% of cases of deaf-mutism are congenital, and approximately 70% of these congenital cases are apparently due to some extraneous factor rather than to heredity. In 1904 Siebenmann,⁶⁰ discussing congenital deaf-mutism, mentioned the possibility of placental infection when the mother was suffering from smallpox or some other infectious disease. The intriguing point is that otherwise, apart from vaguely supported suggestions of fetal meningitis, no definite aetiological agent has ever been named for this, which is the largest group of cases of deaf-mutism; yet it is into this group that our cases fit, and we are able to point with certainty to an infective aetiological agent and to state that in all probability it was rubella.

A number of types of structural defect have been described in the past by various observers. Thus the middle ear may show the greatest change. In other cases there may be gross developmental anomalies of the bony labyrinth or of the membranous labyrinth and specialized epithelial parts. The commonest type of congenital anomaly is that in which, while the middle ear and the bony form of the inner ear are normal, and the vestibule and semicircular canals are more or less normally developed, the cochlea, especially Corti's organ, and the sacculi show evidence of maldevelopment; that is, the specialized epithelial structures which have to do with hearing are poorly differentiated. In this type the hearing is very poor, but deafness is often not complete, while the vestibular apparatus reacts to stimulation. This form of deafness, which is named Scheib's type, or sacculo-cochlear degeneration, was found by Alexander to embrace 70% of cases of congenital deaf-mutism. Fraser also found this type in approximately 70% of congenital cases in the Edinburgh Special School. I believe that it is to this group that we may attach these congenitally deaf-mute infants whose deficiency is the result of maternal rubella, for it is found that our patients are very deaf indeed, but they are not completely insensitive to sounds, and so far as they have been tested they give evidence of an active though reduced vestibular apparatus.

Under one column in Table I, in which are grouped the salient features of my seventeen cases, I have listed my observations of the hearing capacity. It will be seen that many of the children give some evidence of hearing over the tone range 512 to 2,048. Testing is at first extremely difficult, however, as in the main hearing power is so poor that the very young child is little interested in listening. Obviously the children will need to be tested at intervals as they grow up.

I have been able to test the caloric labyrinthine reactions in nine of my seventeen cases. In most of the cases I have noticed that the response is a little reduced when compared

with the normal. In only one case—Case XVII—did I find an ear from which no actual nystagmus was able to be induced, but even in this case it was in only one ear that there was no response. One other interesting point is the absence of any upset to the children from caloric stimulation; none of the "rubella" group were made sick even

when pronounced nystagmus was induced. I do not believe that the utricle and semicircular canals are left untouched by the maternal infection—indeed, damage is evident from the fact that they are less readily excited than normal. By contrast with the gross inactivity of the cochlear or hearing portion of the inner ear, however, it is obvious that often the semicircular canals are largely spared. While I have compared these observations with the well recognized type and largest group of congenital deaf-mute cases—namely, the sacculo-cochlear type—I do so with open-minded speculation that the same or a similar infection may have existed in the past, or that it is simply that the cochlea is more prone to be damaged during fetal life than is the vestibular portion of the inner ear and that such damage may result from a variety of causes.

Associated Defects in the Cases of Deaf-Mutism.

Not one of my seventeen patients has any evidence of cataract; one has had lenses supplied for correction of strabismus. I think this absence of ocular defects is explicable by the fact that the maternal infection did not occur early enough. I am anxious to hear, however, just what number of children with affected eyes will turn out to be also deaf mutes. My belief is that the earlier the infection, the more diverse the damage, or conversely, the later the infection, the greater the hope that some early differentiat-

ing parts such as the eyes may escape.

I have not had opportunity yet to have an expert examine the heart in all my cases. I myself have not detected evidence of patent *ductus arteriosus* or other such defect in any instance. Stunting of growth is evident in eleven of the seventeen cases and obvious microcephaly in three,

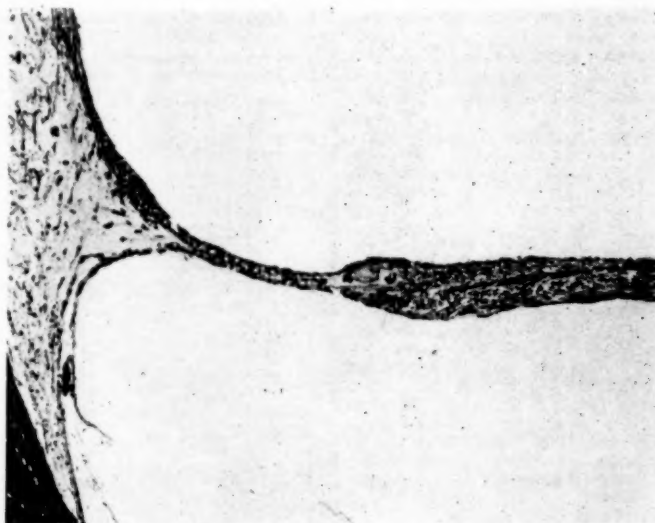


FIGURE I.

Photomicrograph of section through the first turn of the cochlea (x118). There is no evidence of formation of the organ of Corti. The *membrana tectoria* is rudimentary. *Reisner's* membrane is not to be found, unless it be that it remains collapsed on to the *membrana tectoria* and *stria vascularis*. The *stria vascularis* is rudimentary and may not have secreted endolymph at all. The connective tissue structures appear to be more or less normally developed.

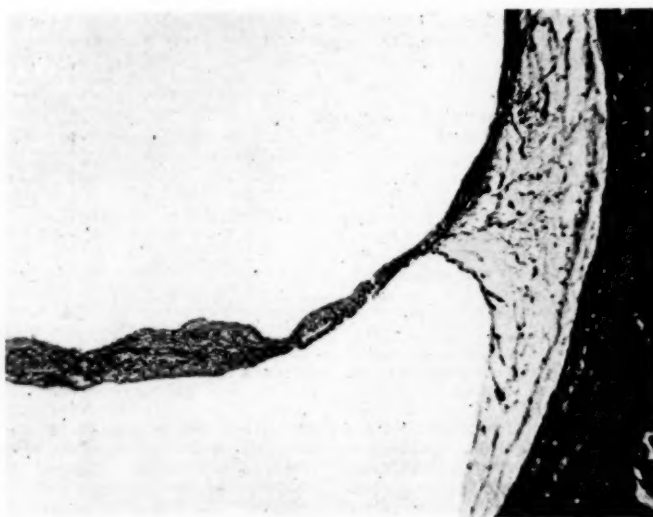


FIGURE II.

See legend to Figure I.

although more children have small heads proportionate to their small stature.

Progress of the Children as they Grow Older.

The oldest of the children is now six and a half years old (Case XVI), another is five and a half (Case I). The youngest has reached three and a half years. I have had opportunity to see the children at intervals during two or three years of their lives, and I have tested their hearing with the audiometer. It is still difficult to forecast the ultimate outcome of their affliction. I shall discuss only their hearing and progress in speech, although I have indicated some details as to growth also in Table I. Several of the children at the age of about three and a half years have commenced to use their hearing. I find it hard to believe that there has been an actual development of the receptive function of the inner ear; rather do I think that the children have come to take some interest in their residual powers as a result of mental growth and training. I am sure that they are still very deaf. Some of these children, with perceptive power in small degree extending over the necessary range for speech perception—512 to 2,048 cycles—may ultimately be able to make use of these islands of perception in order to facilitate education with the assistance of a hearing aid, and they may perhaps develop a more nearly natural

learn, from the voice's qualities, to appreciate the meaning of many of the sounds uttered by the child, in order that such use of the voice will be preserved, for there is thus a greater hope that the child will learn to form intelligible words and to speak with a nearly natural voice later on. While details and methods should be devised by the expert

teacher, I feel that interest in listening and in attempting to form simple words from visual example should be commenced and persevered with from the earliest. In the main, special education by visual methods, with and without sound amplification, will be essential for these children. I suspect that when they are a little older, a poor intelligence quotient may be found in some of the stunted and microcephalic children. Some may be normal; for them special schooling may bring about ability to speak and they may receive an education of fair standard. It is to be realized, however, that the training and teaching of a deaf-mute child are extremely slow and difficult, even when the pupil is of

normal intelligence. We may thus hold out some hope of amelioration of the lot of these children. That an infection can occur and at a stroke can leave so vast a number of children afflicted for life demands that efforts shall

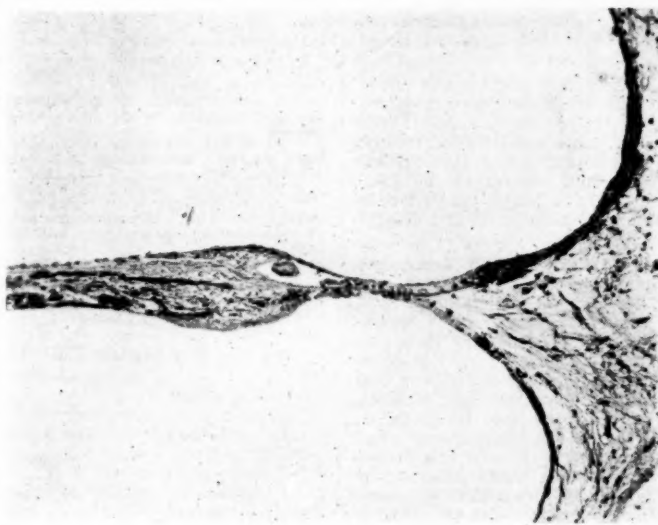


FIGURE III.
See legend to Figure I.

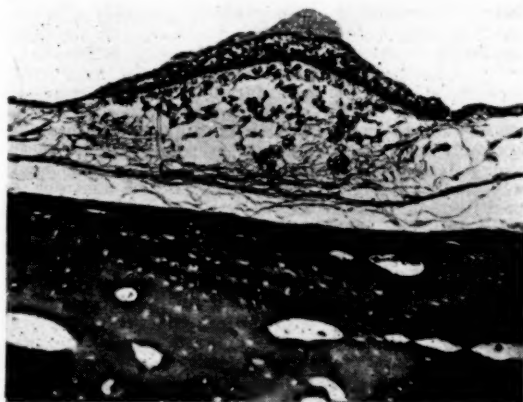


FIGURE IV.

Photomicrograph of section through the ampulla and crista of the right lateral semicircular canal ($\times 118$). Although the general form of the crista is evident, there is poor differentiation of the neuroepithelial cells.

speaking voice. Most of them have a voice and use it to make sounds. I have been at pains to impress upon the parents the point stressed by the Ewings,⁽⁶⁾ of the Manchester School for the Deaf and Dumb, that every effort must be made to preserve the baby voice until such time as special schooling is practicable. Parents must



FIGURE V.

Photomicrograph of section through the ampulla and crista of the right lateral semicircular canal ($\times 264$). Although the general form of the crista is evident, there is poor differentiation of the neuroepithelial cells.

continue to be made to identify the virus precisely, and to endeavour to protect the mothers and their offspring by shielding them from infection in the home and by a process of immunization if that is possible.

Pathological Findings in One Case.

I have, through the kindly cooperation of the medical superintendent and the pathology department of the Royal

Alexandra Hospital for Children, been able to secure the temporal bones of an infant who died at the age of six and a half months. The mother suffered from rubella during the first month of pregnancy. The infant had congenital cataracts, patent *ductus arteriosus*, was undersized and was thought to be deaf. She had been difficult to feed, had suffered from pneumonia two months before death, and died within twenty-four hours of subsequent admission to hospital. A brief report on the aural structures is perhaps relevant to this paper.

The general form of the middle ear, ossicles and inner ear appeared to be normal. Serial sections were prepared at the Kanematsu Institute, Sydney Hospital. With the cooperation of the director of the institute, Dr. A. J. Canny, I have endeavoured to interpret the defects of development in the inner ear. The outstanding feature is the total absence, in both ears, of any differentiation of the primitive cells to form the organ of Corti (Figures I, II and III).

The eighth nerve and spiral ganglion appear to be well formed. The bony spiral lamina and basilar membrane can be readily demonstrated. The *stria vascularis* contains relatively few blood vessels and is shallower and less cellular than usual. The *membrana tectoria* is frequently to be discerned, but it is of rudimentary form and is in most sections surrounded by a layer of nucleated flat cells. Reisner's membrane I have not been able to find at all. The semicircular canals, bony and membranous, are well formed. I have not yet been able to study all sections sufficiently to state whether the receptor end-organ called the crista is sufficiently formed even to be identifiable in each of the canals. That of the right external or horizontal canal, however, is clearly identifiable (Figures IV, V and VI). In this end-organ, too, as in the case of

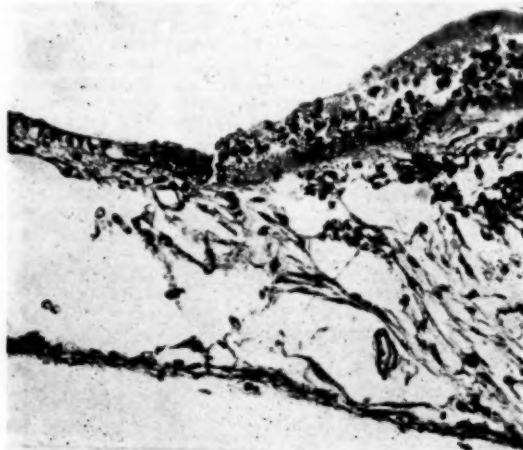


FIGURE VI.

Photomicrograph of section through the ampulla and crista of the right lateral semicircular canal ($\times 264$). Although the general form of the crista is evident, there is poor differentiation of the neuroepithelial cells, except in one small area at the periphery of the crista, where typical columnar epithelial cells are visible.

Corti's organ in the cochlea, cell differentiation into the specialized epithelial formation normally visible, is wanting, except in one small area. This finding is rather contradictory to my observation of the comparatively normal responses to labyrinthine stimulation of older children. I think that the only explanation is that the mother was infected early during the life of the fœtus, with the result that interference with developmental cell differentiation was widespread; this we see from the multiplicity of gross defects observed in this infant even before death, for the mother had suffered from rubella within the first month of her pregnancy, and the baby herself had bilateral cataracts and congenital heart defects and was stunted and extremely difficult to feed.

References.

- ⁽¹⁾ N. McAllister Gregg: "Congenital Cataract following German Measles in the Mother", *Transactions of the Ophthalmological Society of Australia (British Medical Association)*, Volume III, 1941, page 35.
- ⁽²⁾ C. Swan, A. L. Tostevin, the late B. Moore, H. Mayo and G. H. Barham Black: "Congenital Defects in Infants following Infectious Diseases during Pregnancy", *THE MEDICAL JOURNAL OF AUSTRALIA*, September 11, 1943, page 201.
- ⁽³⁾ C. Swan, A. L. Tostevin, H. Mayo and G. H. Barham Black: "Further Observations on Congenital Defects in Infants following Infectious Diseases during Pregnancy with Special Reference to Rubella", *THE MEDICAL JOURNAL OF AUSTRALIA*, May 6, 1944, page 409.
- ⁽⁴⁾ J. Kerr Love: "Deaf Mutism: A Critical Review", *The Journal of Laryngology and Otology*, Volume XXXVI, 1921, page 29.
- ⁽⁵⁾ J. S. Fraser: "The Pathological and Clinical Aspects of Deaf Mutism", *The Journal of Laryngology and Otology*, Volume XXXVII, 1922, pages 13, 57, 126.
- ⁽⁶⁾ Siebenmann: Quoted by Fraser, *loc. cit.*
- ⁽⁷⁾ Alexander: Quoted by Fraser, *loc. cit.*
- ⁽⁸⁾ I. R. Ewing: "Deafness in Infancy and Early Childhood", *The Journal of Laryngology and Otology*, Volume LVIII, 1943, page 137.

THE INFLUENCE OF MALARIA ON THE KLINE AND COMPLEMENT FIXATION (WASSERMANN) TESTS FOR SYPHILIS.

By C. B. Cox,

Major, Australian Army Medical Corps,

AND

M. J. DURANT,

Lieutenant, Australian Army Medical Corps.

(From an Australian General Hospital.)

It has long been recognized that the serological tests used for the diagnosis of syphilis may not be true antigen-antibody reactions, and may therefore give rise to confusing results in diseases other than syphilis. Of these, malaria is one of the best known, and since large-scale military operations have been carried out in malarious areas, the differentiation of reactions due to syphilis and those attributable to malaria ("false positives") has become a matter of considerable importance.

The literature on the subject of non-specific serological reactions in malaria indicates considerable differences of opinion regarding their frequency and their interpretation. Iyengar,⁽¹⁾ in India, after examining 98 cases, concludes that malaria is not a cause of a positive reaction to the Wassermann test, and Pratt Johnson,⁽²⁾ in South Africa, employing four modifications of the same test, states that his investigations indicate that the blood in active benign tertian, malignant tertian and mixed malaria does not give a positive reaction to the Wassermann test. The latter author did find that 20% of sera gave one or more positive reactions in carrying out 738 examinations at intervals on a total of 74 individuals, and 7% of these on being retested gave further positive results; but he believes it reasonable to assume that this small number of cases was due to latent syphilis. Altounyan,⁽³⁾ in Syria, found 11% strongly positive and 12% weakly positive Wassermann reactions in 135 cases of chronic malaria, while in 21 cases of acute malaria the percentages were 9.5 and 30 respectively; but in view of the fact that 13.5% of the serum of 1,263 persons "not suffering from acute malaria or exhibiting gross signs of chronic malarial infection" gave strongly positive reactions and 9.1% gave weakly positive reactions, he concludes that chronic malaria has no appreciable effect on the Wassermann reaction, although acute malaria has an undoubted effect in increasing the anticomplementary powers of the serum.

Kolmer stated in 1929⁽⁴⁾ that it appeared to him improbable that malaria parasites *per se* could produce the reagins responsible for Wassermann reactions; yet in a study of various serodagnostic tests for syphilis, conducted in 1935 by the Public Health Service of the United

States of America,⁽⁵⁾ Kolmer's laboratory reported seven "false positives" in 36 specimens of blood taken from patients suffering from uncomplicated malaria. Other well-known serologists, using methods in which they were expert, also returned "false positive" or "doubtful" results, and among the total of 457 tests on specimens taken from the 36 malarial donors, there were 66 (14.4%) positive reactions and a further 6.1% "doubtful" reactions. Discussing these findings, Hazen *et alii*⁽⁶⁾ point out that for only 14 of the 36 patients was there complete accord among the results obtained by the 13 serologists taking part in the study. These authors discuss also the results of an additional study on 266 patients, with four tests in most cases, the tests being carried out by Hinton, Kline (using both his diagnostic and exclusion tests) and Kolmer. The percentage of positive results was not so high as it was in the first study; but they consider it higher than that to be expected among white subjects when care has been taken to exclude syphilis. Further, they discuss results published by serologists up to that time, and in conclusion make the following statement:

It seems safe to assume that malaria, like leprosy, can be the cause of positive serologic reactions to tests for syphilis. It is probable that more than one patient has been placed under treatment for syphilis when malaria was the cause of his positive serologic reaction.

More recently, Kitchen *et alii*⁽⁷⁾ have made a comprehensive contribution to the subject by utilizing the opportunity presented by a decision to administer malarial therapy to a group of non-syphilitic patients with functional psychoses. Kitchen and his co-workers studied the Wassermann and Kahn reactions before, during and after twenty-five naturally induced attacks of malaria in these patients. They obtained positive reactions at some stage in every case in which malaria developed clinically, but in two cases no positive results were obtained from the complement fixation test, and in two others no positive Kahn reactions were obtained. Of the positive reactions, 72% made their first appearance during the third and fourth weeks followed inoculation. The first positive reactions were observed both before and after the period of clinical activity in a few instances; but in 68% of the attacks these reactions occurred within the first two weeks of the febrile period. The duration of the "seropositive" period exceeded three weeks in 60% of the cases and extended beyond four weeks in 48%. *Plasmodium vivax* infections tended to induce a greater proportion of positive reactions than *Plasmodium falciparum* infections. Positive reactions were relatively higher among females than among males, and among persons up to thirty-five years of age than among those over that age. The percentage of positive reactions was highest during the period from fifteen to twenty-one days after the last previous paroxysm.

The evidence that malaria *per se* can give rise to non-specific positive reactions with the commonly used serological tests for syphilis seems conclusive, and is in accord with recent experience of pathologists in this country dealing with service personnel who have served in malarious areas.

In order to distinguish between specific and non-specific reactions, a method has been described recently by Kahn *et alii*⁽⁸⁾ having for its basis observations that reacting serum of syphilitic patients shows stronger reactions at 37° C. than at 1° C., whereas reacting serum of non-syphilitics shows the opposite. He has, therefore, introduced the Kahn verification test as a supplementary procedure to classify reactions as syphilitic or non-specific. Some workers, for example Beveridge,⁽⁹⁾ have reported this verification test to be of value; yet it does not entirely exclude the general biological (non-specific) type of reaction, since Beveridge found 14 positive reactions with serum from presumably non-syphilitic subjects in a total of 335 specimens examined. Moreover, the test is inconclusive when only a small number of specimens of serum is involved.

The Practical Problem.

From an analysis of the results of the quoted authors, it might appear that a person who was suffering or had suffered from malaria, ran a considerable risk of being

falsely classed as syphilitic. However, our experience in the laboratory of an Australian special hospital in Palestine in 1941-1942 with tests on about 8,000 specimens of serum, a proportion from soldiers with a history of malaria, suggested to us that this unfortunate circumstance should be one of great rarity. Firstly, the fact that a diagnosis of syphilis is primarily a problem for the clinician should be again emphasized, for there still exists a tendency to rely entirely on the report of the serologist, even though he may have no personal knowledge of the patient, nor even be supplied with a helpful history. The task of the serologist is to carry out a test for syphilitic reagin, not to diagnose syphilis; yet even this task is complicated by reason of ignorance concerning the nature of this reagin and a lack of knowledge as to whether or not the same substance can exist sometimes in serum of normal humans, for it does appear to be present normally in serum from many animals.

Secondly, it appears relevant to point out that some of the quoted authors conducted their examinations from a purely experimental point of view, in a manner bearing little relationship to the actual conditions met with in practice. For example, the work of Kitchen and his colleagues⁽⁷⁾ was done on patients who were permitted to have repeated attacks of malaria without treatment, in some cases lasting over seventy days. Such cases should be encountered but rarely in ordinary practice.

Experimental Investigation.

In order to determine the likelihood that the occurrence of serological reactions in malaria patients would lead to a false diagnosis of syphilis, we have carried out both flocculation and complement fixation tests on a number of soldiers suffering from acute benign tertian malaria, and also on a number of treated convalescents ten to fourteen days after parasites had been demonstrated in their blood. In no case was there any evidence of coexistent syphilis or any history of the disease. Specimens of venous blood were collected from 175 patients suffering from malaria, usually within thirty minutes of their admission to hospital, and a record was made of the temperature, date of first attack and number of relapses. We were able to collect second specimens of blood from 83 of these patients toward the end of their treatment—that is, ten to fourteen days after the diagnosis had been made—at which time no parasites were found in the peripheral blood and the patients were afebrile. An additional 57 convalescent patients were examined similarly, but we had been unable to collect blood from these during the febrile stage.

Tests Employed.

Two tests were carried out on each specimen of serum—namely, the Kline diagnostic test and a complement fixation test. The technique of the former was essentially as described by Kline,⁽¹⁰⁾ antigen manufactured by the Commonwealth Serum Laboratories being used. Results are reported on the size of the clumps, viewed microscopically, in terms of "pluses", thus: "±" = indefinite aggregation (doubtful); "+" = small clumps (weak positive); "++" = clumps of moderate size (intermediate positive); "+++" = large clumps (strong positive).

The complement fixation technique which we employed has proved both sensitive and specific in our hands; but it seems unnecessary to describe it in detail, since modifications of the original Wassermann test are already numerous, and there seems no occasion to overburden the literature on the subject any further. In brief, the method requires four tubes for each specimen of serum, with 0.1 cubic centimetre in the serum control, and 0.1 cubic centimetre, 0.05 cubic centimetre and 0.025 cubic centimetre in the other three tubes. Two units of complement are used in the serum control and 2.5 units in the remaining tubes. The antigen is a cholesterolized alcoholic extract of ox heart, highly concentrated and diluted to one in 200 before use. The volume in each tube at the fixation stage is 0.75 cubic centimetre and at the hæmolytic stage one cubic centimetre. Fixation is allowed to take place at approximately 6° C. for eighteen hours; this is

followed by ten minutes in the water-bath at 37° C. After addition of the sensitized sheep cells, readings are made each fifteen minutes, that at the end of one hour being regarded as final, provided, of course, that the complement-antigen controls are satisfactory. In the reporting of results, the following terms are used: negative—all four tubes show complete hæmolysis; complete positive ("C.P.")—tube I (serum control) shows complete hæmolysis and tubes II, III and IV show no hæmolysis; incomplete positive, strong ("I.C.P. Strong")—tube I shows complete hæmolysis, tube II no hæmolysis and tubes III and IV some hæmolysis, usually more pronounced in tube IV; incomplete positive, weak ("I.C.P. Weak")—tube I shows complete hæmolysis and tubes II, III and IV some hæmolysis, which may be complete in tubes III and IV; anticomplementary ("A.C.")—there is no hæmolysis or incomplete hæmolysis in tube I, and similar lack of hæmolysis is found in one or more of the successive tubes.

Another type of reaction is sometimes seen—namely, the so-called prezone reaction—in which the serum control tube together with the tube containing the largest amount of test serum shows complete hæmolysis, but partial inhibition of hæmolysis is found in the tubes containing the lesser amounts. The prezone type of reaction appears to be due to a supersensitivity of complement to antigen. Normally, it has caused us no difficulty; but with the serum from malaria patients we have found a tendency for this type of reaction to occur. Prezone reactions have not been recorded separately, being regarded as "negative".

The Value of Two Concurrent Tests.

Despite the large number of techniques available for the serological diagnosis of syphilis, some bearing the names of workers who have devised them, the fact is that all may be grouped as either flocculation tests or complement fixation tests, the latter being known commonly as Wassermann tests.

In general terms, it may be stated that the flocculation test, typified by the Kline test, is the more sensitive, while the complement fixation test is the more specific. Therefore, we consider that for the serological diagnosis of syphilis the combination of a flocculation test and a complement fixation test is an advisable procedure, because the former should ensure that no positive result is overlooked, and although it may produce some non-specific reactions, these should be excluded by the complement fixation test. Of the flocculation tests available the Kline diagnostic test has the advantages of rapidity and ease in manipulation. In untreated secondary and tertiary syphilis one almost invariably obtains a strongly positive response to the Kline test and a completely positive response to the Wassermann test. After treatment the two results may not run parallel, and we have noted that the Kline reaction tends to remain positive after the

same serum fails to react to the Wassermann test. In primary syphilis, when *Treponema pallidum* has been found in the lesion, both serological tests may give negative results or both may give positive results; but sometimes the Kline test produces a reaction when no reaction is given to the Wassermann test. On the other hand, we have noted in a few cases of late congenital syphilis that the Kline test has produced only a weak reaction, while the complement fixation test produced a strongly positive reaction. To sum up, we are of the opinion that the occurrence of reactions other than a combination of two strong positives should not be regarded as even *prima facie* evidence of syphilis in the absence of other confirmatory evidence—for example, demonstration of the *Treponema pallidum* in early infections or stigmata of the disease in later cases.

Results of Serological Tests on Patients Harboring Benign Tertian Malaria Parasites.

The results with serum from patients suffering from acute malaria are set out in Table I, together with the number of confirmed and treated attacks from which these men had suffered previously.

In nine cases the number of relapses could not be exactly ascertained and these have been noted merely as "several" attacks. It will be seen that "doubtful" and "positive" reactions to the Kline test were obtained, amounting to 17 in a total of 175 specimens of serum examined; but positive reactions to the Wassermann test numbered only four, all being weak. The point of greatest interest, however, is that in only one case did the results of the two tests run parallel. In this single instance neither test produced a strongly positive result. The response to the Kline test was reported as "++" and that to the Wassermann test as "incomplete positive weak"; this combination we should not regard as indicative of syphilitic reagin, except in the case of known syphilis under treatment. In the absence of knowledge about such a case we should request a repeat examination with fresh serum, and actually when this procedure was adopted one week later the response to the Wassermann test had become negative, while that to the Kline test remained "++", as previously. We then had no doubt that the result obtained with the Kline test represented a general biological reaction and not one due to syphilitic reagin, although we were unable to follow the subject with further blood tests.

Another feature brought out in the table is the relative frequency of anticomplementary reactions obtained with the Wassermann test, and also the number of specimens of serum, shown by the figures in parentheses, in which hæmolysis was slow to take place. By slow hæmolysis we mean that the process was not complete in fifteen minutes as is usual with normal serum, yet was complete at one hour when final readings were made, so that these results were reported as "negative".

TABLE I.
Benign Tertian Malaria Parasites Present in Blood.

Number of Confirmed Attacks.	Total Cases	Results of Kline Test.					Results of Wassermann Test.					Results of Both Tests.		
		Neg- ative.	Doubt- ful.	"+"	"++"	"+++"	Neg- ative.	¹ Neg- ative ("S.H.")	"A.C."	"I.C.P. Weak."	"I.C.P. Strong."	"C.P."	Weak Positive.	Strong Positive.
Primary attack	27	25	—	1	—	1	25	(2)	2	—	—	—	—	—
Two	15	15	—	—	—	—	15	—	—	—	—	—	—	—
Three	17	16	1	—	—	—	16	(1)	1	—	—	—	—	—
Four	20	17	1	1	—	1	19	(2)	1	—	—	—	—	—
Five	19	18	—	1	—	—	16	(1)	3	—	—	—	—	—
Six	18	16	1	—	1	—	16	(1)	1	1	—	—	1	—
Seven	18	15	2	—	1	—	16	—	1	1	—	—	—	—
Eight	11	10	—	1	—	—	11	—	—	—	—	—	—	—
Nine	9	6	—	2	1	—	8	—	1	—	—	—	—	—
Ten	12	2	—	—	—	—	2	—	—	—	—	—	—	—
Eleven or more	10	9	1	—	—	—	9	—	1	—	—	—	—	—
"Several"	9	9	—	—	—	—	8	—	1	—	—	—	—	—
Grand total	175	158	6	6	3	2	161	(7)	10	4	—	—	1	—

¹ Negative ("S.H.") = result negative, but hæmolysis of the red cells was slower than with the majority of non-reacting specimens. For other abbreviations used, see text.

TABLE II.
No Malaria Parasites Found in Blood and Patients Afebrile.

Number of Confirmed Attacks.	Total Cases	Results of Kline Test.					Results of Wassermann Test.					Results of Both Tests.		
		Neg- ative.	Doubt- ful.	" + "	" + + "	" + + + "	Neg- ative.	¹ Neg- ative ("S.H.")	"A.C."	"I.C.P. Weak."	"I.C.P. Strong."	"C.P."	Weak Positive.	Strong Positive.
Primary attack ..	32	21	—	5	4	2	29	(1)	2	1	—	—	1	—
Two	4	4	—	—	—	—	4	—	—	—	—	—	—	—
Three	7	5	—	1	—	1	7	—	—	—	—	—	—	—
Four	21	19	1	—	—	1	21	—	—	—	—	—	—	—
Five	19	19	—	—	—	—	19	(1)	—	—	—	—	—	—
Six	13	11	—	1	1	—	13	—	—	—	—	—	—	—
Seven	14	11	1	1	—	1	11	—	1	—	—	2	1	1
Eight	12	12	—	—	—	—	12	—	—	—	—	—	—	—
Nine	7	5	—	1	1	—	7	—	—	—	—	—	—	—
Ten	2	2	—	—	—	—	2	—	—	—	—	—	—	—
Eleven or more ..	9	9	—	—	—	—	9	—	—	—	—	—	—	—
Grand total ..	140	118	2	9	6	5	134	(2)	3	1	—	2	2	1

¹ Negative ("S.H.") = result negative, but hemolysis of the red cells was slower than with the majority of non-reacting specimens. For other abbreviations used, see text.

Results of Serological Tests on Patients during Convalescence.

It is not usual for a clinician to request the examination of a patient's serum for syphilitic reagin during a febrile period resulting from a known infectious disease such as malaria, so it becomes of more importance to see the effect on the serological reactions of blood collected from patients during the convalescent stage, when other investigations may be indicated. Such examinations have been made in 140 cases, 83 of these from cases included in Table I. The results in this series are incorporated in Table II.

It will be noted that the number of positive Kline reactions has become rather greater, and the tendency is for the reactions to be stronger. On the other hand, the Wassermann tests produce fewer anticomplementary and fewer positive results. In only three cases did the tests give parallel results, and in only one of these were both reactions strongly positive (Kline, "+++"; Wassermann, "complete positive"). However, this particular patient had been examined while febrile eleven days previously, and at that time both tests produced negative results, so the results in convalescence appeared to be "false positives". Nevertheless, he was observed over a period of weeks and eventually his serum failed to react to either test. No history suggestive of syphilitic infection was elicited, nor were any signs of the disease found. With regard to the other two cases, in the first the reported results were Kline "+" and Wassermann "C.P." (both tests produced negative results when the patient was febrile), and in the second case the results were Kline "+" and Wassermann "I.C.P. weak".

In Table III the percentages of "negative", "positive" and "doubtful" results have been brought together for purposes of comparison of both series.

TABLE III.

Results of Tests.	Malaria Parasites Present.	No Malaria Parasites Found; Patients Afebrile.
Kline test:		
Negative	90.3%	84.3%
Doubtful and weakly positive ("+" or "++") ..	8.6%	12.1%
Strongly positive	1.1%	3.6%
Wassermann test:		
Negative	92.0%	95.7%
Anticomplementary	5.7%	2.1%
Weakly positive	2.3%	0.7%
Strongly positive	0	1.5%

From Tables I and II no correlation between the number of relapses and the number of positive reactions is to be seen, although the series are comparatively small and definite conclusions cannot be drawn.

Effect of Temperature on the Serological Reactions.

In the acute cases it seemed of interest to determine also whether temperature had any bearing on the frequency of positive reactions, but no obvious relationship is apparent in Table IV.

Combinations of Reactions Found.

In Table V are set out the types of reactions found in the 82 cases investigated during both the acute and the convalescent stages. For simplicity "+" means here a reaction to either Kline or Wassermann test of weak or strong character; but "+" has been retained for the "doubtful" Kline result, and "A.C." indicates "serum anticomplementary" in the Wassermann test.

TABLE IV.
Malaria Parasites Present. Comparison of Patients' Temperatures with Serological Findings.

Temperature. (Degrees Fahrenheit.)	Total Cases.	Results of Kline Test.				Results of Wassermann Test.				Results of Both Tests.		
		Negative.	Doubtful.	"+" or "++"	"+++"	Negative.	"A.C."	"I.C.P."	"C.P."	Weak Positive.	Strong Positive.	Kline, Positive; Wassermann, "A.C."
98 or below ..	7	6	—	—	1	6	1	—	—	—	—	1
98.2 to 99 ..	12	11	—	—	1	12	—	—	—	—	—	—
99.2 to 100 ..	39	35	2	2	—	36	2	1	—	—	—	1
100.2 to 101 ..	36	34	1	1	—	34	1	1	—	—	—	—
101.2 to 102 ..	52	46	2	4	—	45	5	2	—	—	—	—
102.2 to 103 ..	22	20	1	1	—	21	1	—	—	—	—	—
Over 103 ..	7	6	—	1	—	7	—	—	—	—	—	—
Grand totals ..	175	158	6	9	2	161	10	4	—	1	—	2

TABLE V.
Comparisons of Serological Reactions Obtained in 53 Cases.¹

Number of Cases.	Parasites Present.		No Parasites Present.	
	Results of Kline Test.	Results of Wassermann Test.	Results of Kline Test.	Results of Wassermann Test.
64	—	—	—	—
2	+ or ±	—	—	—
1	—	+	—	—
3	—	A.C.	—	—
1	±	+	—	—
2	—	—	+	+
1	—	—	—	A.C.
6	+ or ±	—	+ or ±	—
1	+	+	—	—
1	+	A.C.	+	—
1	+	—	±	A.C.
0	+	+	+	+

¹ Explanation of abbreviations:—Kline test: "±" = doubtful, "+" = weak or strong positive; Wassermann test reaction: "A.C." = anticomplementary, "—" = weak or strong incomplete positive, or complete positive.

The interesting feature to be seen here is that in no case did both tests produce positive results in both the acute and the convalescent stages. In a larger series it seems reasonable to assume that this combination might be encountered; but it should not arise often, and a history of malaria would then suggest caution in reporting the result without repeated blood tests at intervals.

Differences between Immediate and Delayed Separation of Serum.

From 25 patients we collected blood in two portions, separating serum from the one without delay and from the other after it had stood in contact with the clot in a refrigerator for four or five days. We found that serum removed after standing tended to give an anticomplementary Wassermann reaction more readily than freshly separated serum; but this difference was not more pronounced than with normal serum. Nevertheless, in view of the fact that anticomplementary reactions appear to be relatively common in malaria, it should be the aim to separate serum from clot as soon as possible after collection of the blood specimen in order to minimize the chance of occurrence of this type of reaction. The effect of delayed separation on the Kline test was negligible.

The Differentiation of Positive Results due to Malaria and to Syphilis.

Fischer and Günsberger⁽¹⁰⁾ have drawn attention to the following facts: firstly, every attack of malaria is accompanied by destruction of a number of erythrocytes; and, secondly, extracts from blood-containing organs are used as antigens in the serological diagnosis of syphilis. These authors make the assumption that reactions to the Wassermann test applied to malaria serum are brought about by the reaction of erythrocyte lipoids in the antigen with homologous antibodies in the serum. They describe an extract of ox heart, relatively poor in erythrocyte lipoids, which, while a good syphilitic antigen, reacts only slightly in malaria, and also an extract of erythrocyte lipoids which they claim reacts with malarial serum and not with syphilitic serum. By using both extracts they claim that they further are able to differentiate between positive Wassermann reactions due to malaria and those due to syphilis.

We have carried out a small number of tests with both types of serum, employing the antigens prepared as described by these authors, but we have been unable to confirm their results. In our experience the lipid extract they describe for use in the diagnosis of malaria is no more than an inferior syphilitic antigen. Thus in one series consisting of blood from eight febrile malaria patients, two normal persons and two known syphilitics, all specimens gave clear negative results with the exception of the last two, which gave weakly positive results. These specimens of serum gave strongly positive results with the

antigen we usually employ. Similar results were given in other comparable series. We are of the opinion, therefore, that the differentiation of the positive Wassermann reaction due to malaria from the true syphilis reaction is not possible by means of the method described by these authors.

Conclusions.

Although we realize that the number of examinations reported here is relatively few, we consider that the results indicate little likelihood of branding a patient with a history of malaria as syphilitic, provided the criteria to which we have drawn attention are invariably kept in mind. A history of malaria associated with positive results to serological tests, particularly when flocculation and complement fixation methods lack agreement, indicates to us that both tests should be repeated with a fresh specimen of serum before a report is made to the clinician. By the adoption of this procedure the nature of the reactions (that is, whether they are specific or non-specific) should be revealed. In every case, with the exception of treated or previously diagnosed syphilis, in which we fail to find correspondence between the results of the two tests, we do not report our findings, but request fresh serum in a week or fortnight, whichever is the more convenient, and we suggest that this procedure be adopted by pathologists as a working rule. We regard this as better than reporting the results of multiple tests which do not agree, because such a report serves merely to confuse a clinician. If repeated tests continued to reveal discrepancies and definite signs of syphilis are lacking, we have no hesitation in regarding the serological results as "false positives" and in informing the clinician that we regard them as such. At the same time it must be made clear that we have found that this circumstance arises but seldom in practice.

Summary.

1. Kline and complement fixation (Wassermann) tests for syphilis have been carried out on 175 specimens of serum from non-syphilitic patients suffering from acute malaria and on 140 specimens of serum from non-syphilitic convalescent malaria patients.
2. A proportion of "false positive" results was obtained with each test in both series.
3. In acute malaria the observed positive reactions were generally weak, and only one specimen of serum reacted similarly to both tests. The complement fixation test produced a relatively high percentage of anticomplementary reactions.
4. During convalescence from malaria the Kline test produced a slightly higher proportion of "false positive" results than in the acute stage, whereas the reverse was the case with the complement fixation test, and anticomplementary reactions were also fewer.
5. Correspondence in serological reactions was not seen with serum tested during both the acute and the convalescent stages, and the conclusion is drawn that little risk of branding a malaria patient as syphilitic should occur if repeated examinations of his serum are made at intervals, both a flocculation test and a complement fixation test being employed.
6. No relationship between the occurrence of "false positive" results and the number of relapses was found, and in acute malaria there appeared to be no relationship between the patients' temperatures and "false positive" results.
7. We have been unable to differentiate the "false positive" reaction in malaria from the specific reaction in syphilis by means of the erythrocyte lipid extract described by Fischer and Günsberger.

Acknowledgement.

Our thanks are due to Major-General S. R. Burston, Director-General of Medical Services, for permission to publish this paper.

References.

- ⁽¹⁾ R. R. K. Iyengar: "Significance and Value of a Positive Wassermann Reaction in Malaria", *Indian Journal of Medical Research*, Volume VIII, 1920, page 136.
- ⁽²⁾ J. Pratt Johnson: "The Diagnosis of Syphilis in Malarial Subjects by the Wassermann Reaction", *The Journal of Pathology and Bacteriology*, Volume XXIV, 1921, page 145.
- ⁽³⁾ C. H. R. Altounyan: "Serum Diagnosis in Syria", *The Lancet*, Volume I, January 12, 1924, page 73.
- ⁽⁴⁾ J. H. Kolmer: "Truths about the Serum Diagnosis of Syphilis", *The Journal of the American Medical Association*, Volume XCIII, 1929, page 1429.
- ⁽⁵⁾ United States Public Health Service: "The Evaluation of Serodiagnostic Tests for Syphilis in the United States", United States Government Printing Office, Washington, 1935, page 10.
- ⁽⁶⁾ H. H. Hazen, F. E. Seneat, T. Parran, A. H. Sanford, W. U. Simpson and R. A. Vonderlehr: "Serologic Evidence of Syphilis in Malarial Patients", *Archives of Dermatology and Syphilology*, Volume XXXVII, 1938, page 431.
- ⁽⁷⁾ S. F. Kitchen, E. L. Webb and W. H. Kupper: "The Influence of Malarial Infections on the Wassermann and Kahn Reactions", *The Journal of the American Medical Association*, Volume CXII, 1939, page 1443.
- ⁽⁸⁾ R. D. Kahn, C. B. McDermott and S. Marcus: "Effect of Temperature on Kahn Reaction", Parts I, II, III and IV, *American Journal of Syphilis, Gonorrhea and Venereal Disease*, Volume XXV, 1941, pages 151, 157, 162, 173.
- ⁽⁹⁾ W. J. M. Beveridge: "The Kahn Verification Test: A Preliminary Note", *Edinburgh Medical Journal*, Volume L, June, 1943, page 344.
- ⁽¹⁰⁾ C. Fischer and O. D. Günsberger: "Ueber die Ursache der positiven Wassermann-Reaktion bei Malaria", *Zeitschrift für Immunitätsforschung und experimentelle Therapie*, Volume LXXVIII, 1933, page 295.

Reports of Cases.

ANEURYSM OF THE VENTRICLE OF THE HEART.

By T. J. F. FRANK,
Melbourne.

AN article describing in brief the aetiology, pathology, clinical features, diagnosis and prognosis of aneurysm of the ventricle of the heart was published recently in the *THE MEDICAL JOURNAL OF AUSTRALIA*.⁽¹⁾ In addition, a case recognized in September, 1941, approximately three months after an attack of coronary occlusion, was recorded. The history of this patient had been followed till February, 1943, and in the present paper the subsequent events and post-mortem findings will be described.

Clinical Record.

When the patient was examined in April and September, 1943, the principal symptoms were gradually increasing weakness, more marked dyspnoea, angina of effort on any but the slightest exertion and a liability to attacks of bronchitis. Oedema of the feet had at no time been noted. At the last visit on July 20, 1944, he had recovered from a recent bout of influenza, which had further diminished his cardiac reserve; several days previously a severe mid-epigastric pain of several hours' duration followed walking against a cold wind. On examination of the patient, dyspnoea and cyanosis of the lips and of the ear tips were noted. The apex beat of the heart was palpated in the fifth left intercostal space seven inches (17.5 centimetres) from the mid-sternal line, pulsation was present in the region of the left nipple and of the left anterior axillary line, and a systolic murmur was audible over the apex and in the region of the left nipple. The rhythm was regular. The systolic and diastolic blood pressure readings were respectively 140 and 100 millimetres of mercury. There were no signs of congestive heart failure. The urine contained neither albumin nor sugar.

An X-ray examination of the heart on August 14, 1944, showed no change from the previous findings in September, 1943. The report read as follows: "There is an unusually large aneurysm arising from the anterior wall of the left ventricle and reaching the thoracic wall antero-laterally at the level of the fourth left interspace; the vascular pedicle is slender."

On September 30, 1944, the patient felt fit on retiring to bed after having visited several friends in the afternoon. However, during the night a severe mid-epigastric pain associated with a feeling of suffocation and constriction in the chest awoke him from his sleep; the patient had experienced many similar attacks in the past three years. Death occurred within one hour.

Post-Mortem Findings.

The following findings were noted at a post-mortem examination made on October 2, 1944, approximately thirty-two hours after death.

The body was that of a spare, middle-aged male; gross cyanosis and post-mortem staining were present, but no oedema was noted.

Old fibrous adhesions were present between the two layers of the pericardium over the left ventricle, whilst the epicardium was generally fibrous. The greatly enlarged heart weighed 34 ounces (964 grammes). Both the right ventricle and the atrium were dilated; the muscle of the right ventricle was pale red and firm. The left ventricle was very large and its muscle generally was densely fibrotic. There was a large aneurysm of the anterior wall of the left ventricle projecting upwards and forwards; its orifice was about two inches (five centimetres) and the aneurysm itself approximately three inches (7.5 centimetres) in diameter. The aneurysm was partly filled with old laminated blood clot, whilst its wall was densely fibrous. The left ventricle was greatly dilated, whilst the left atrium was moderately dilated. The heart valves were all normal and the *foramen ovale* was closed. The orifices of the coronary arteries were patent, but throughout the course of these vessels considerable thickening, atheroma and extensive calcification were present; the left descending branch was completely occluded at its origin. The size of the aorta was normal; the arch and the abdominal portion were atheromatous. The renal, splenic, superior mesenteric and cerebral arteries appeared normal, whilst the femoral veins and pulmonary arteries were patent.

The lungs were brownish-red, congested and oedematous. The liver and spleen, both of which were firm and congested, weighed 66 ounces (1,871 grammes) and eight ounces (227 grammes) respectively. The kidneys were fairly large, each weighing six ounces (170 grammes); the capsule was readily stripped, a smooth, red surface being left. Examination of sections revealed that the cortex and medulla were wide and regular and the blood vessels were normal.

A post-mortem diagnosis of atherosclerosis, coronary occlusion, aneurysm of the left ventricle, adherent pericardium and cardiac failure was made. All these features had been recognized during life except for the pericardial lesion. This finding is occasionally noted soon after the onset of an infarct situated near the pericardium by the auscultation of a pericardial friction rub. Moreover, pericardial thickening overlying an aneurysm is a common finding at autopsy. Death resulted from cardiac failure, a sequel of a gradual narrowing of the coronary arteries.

Summary.

1. The final clinical notes of a male patient, aged fifty-seven years, with an aneurysm of the left ventricle of the heart, whose case was previously recorded, are reported, together with detailed post-mortem findings.
2. The aneurysm was a sequel to myocardial infarction following coronary occlusion, a result of atherosclerosis.
3. Cardiac failure, secondary to a progressive obstruction of the coronary arteries by atherosclerosis, was the cause of death.
4. The duration of life from the time of establishment of the diagnosis of cardiac aneurysm was thirty-seven months.

Acknowledgements.

I am indebted to Dr. R. J. Wright-Smith, pathologist to the Royal Melbourne Hospital, for the use of his detailed post-mortem notes, and to Dr. F. Stephens for the numerous X-ray examinations of the chest.

Reference.

- ⁽¹⁾ T. J. F. Frank: "Aneurysm of the Ventricle of the Heart, with the Report of a Case", *THE MEDICAL JOURNAL OF AUSTRALIA*, Volume I, 1944, page 526.

Reviews.

A MANUAL FOR PATIENTS WITH HIGH BLOOD PRESSURE.

IRVINE H. PAGE's manual on hypertension, designed not for physicians but for laymen, is a terse and well-written little book; but two questions arise at once on picking up such a book.¹ The first is whether the author can compress into a brochure of 70 small pages an authoritative account of a subject by no means easy to explain and by no means yet free from obscurity. This question may on the whole be answered in the affirmative. Certain criticisms suggest themselves. References to the rarer clearance tests are of doubtful value, and perhaps the diagrams of arterio-sclerotic changes will alarm lay readers more than the reassuring remarks about fluctuating pressures will comfort them. It is doubtful also whether it is wise to state that possibly five or six light meals a day are better than three or four; it might be argued that patients' inward searchings of mind and spirit are not always soothed by departures from the general pattern of contemporary life. We wonder, too, why it is necessary to mention psycho-analysis. But these are mainly matters of detail. The second question is really the important one, whether it is wise or necessary to write a handbook for laymen on this subject at all. Diabetes is probably the ideal topic for a patients' handbook, for here can be given vital, specific and definite instructions which must be followed day by day if the patient is to prosper. But it is doubtful whether it is wise to put a book on so protean and far-reaching a disease as hypertension into the hands of the usually worrying and anxious sufferers. Nervous patients are likely enough at any time to take unwarranted meanings from perfectly harmless remarks, and one hesitates to present them with accounts of kidney extracts, neurosurgical procedures and even nephrectomy. Perhaps the author feels that the time has come to answer in our own way that flood of medical writing in the lay Press which, in supplying the daily bread of the popular paragraphist, supplies also food for introspection in its readers. We cannot help feeling that with such handbooks as this, accurate though they may be, we may "nurse the pinion which impell'd the steel" and actually encourage an unduly morbid interest in disease. Dr. Page has not convinced us that his book can do more than a fraction of the good that he has done in his personal contacts with sufferers from hypertension. Though he disclaims any design of writing for the medical profession, his book could be read with profit by the doctors themselves. His refutation of the absurd total prohibition of red meat, his sage advice about alcohol and tobacco, his remarks on rest and fatigue, and above all his shrewd analysis of the typical hypertensive's personality, could be read with profit by all. These patients are, as he so truly states, "angry about things which do not concern them and rebellious toward things over which they have no control". But these truths do not convince us that it is wise to introduce such patients to the involved and still obscure pathology and the groping and unsatisfactory treatment of hypertension.

A YEAR BOOK ON MEDICINE

"THE 1944 YEAR BOOK OF GENERAL MEDICINE" has been published² and will be welcomed by physician specialists and general practitioners. It is one of the twelve volumes comprising the "Practical Medicine Series of Year Books" founded in 1900 and published continuously since then.

This year's volume follows the style generally adopted and is divided into five parts. The first part, edited by G. F. Dick, deals with infectious diseases. Special emphasis has been laid on military and tropical medicine. The subjects dealt with include immunization in the United States Army, discussions on several of the diseases, effectiveness of

typhoid vaccine, tsutsugamushi fever, sandfly fever, yaws and the control of the body louse. In regard to the common cold investigations in the use of sulphadiazine are mentioned and the not very enthusiastic conclusion is stated that the drug will be useful to give protection against severe secondary infection. The editor wisely points out that it will be difficult to tell in advance which colds are likely to be followed by severe complications. Patulin is mentioned hopefully, but this product had not then been given its quietus. In regard to malaria, research carried out at Harvard University is mentioned, the routine treatment recommended by the Australian army authorities is detailed and the paper by E. T. Brennan, of Sydney, is abstracted. Typhus and virus diseases are discussed, the latter having a good deal of space devoted to them.

The part on diseases of the chest is edited by J. Burns Amberson and prominence is given to contributions dealing with respiratory function. The pneumonias are considered at some length and particular reference is made to their treatment with the sulphonamides and with penicillin. Rheumatic pneumonitis is mentioned and atypical pneumonia caused by a psittacosis-like virus. A paper on bronchography in unresolved pneumonia by G. S. Grier is abstracted. No less than twenty-five pages are given over to the subject of tuberculosis. This disease is considered from many aspects. Among these may be mentioned prothrombin deficiency and vitamin K, chemotherapy with promin and the determination and treatment of pressure cavities in pulmonary tuberculosis. Neoplasms are dealt with and some ten pages are devoted to trauma; chest wounds and mass asphyxia (the tube shelter disaster) are prominent in this section, and an important contribution on pulmonary oedema due to gas poisoning is abstracted.

The part devoted to diseases of the blood and blood-forming organs and diseases of the kidneys is edited by G. R. Minot and W. B. Castle. One of the most important sections, as might be expected, is that dealing with transfusions of blood and blood substitutes. Then are considered in turn the anemias, polycythemia, infectious mononucleosis, agranulocytosis, the leuchemias, purpura and telangiectasia, hæmophilia and prothrombin deficiency. The section on infectious mononucleosis is particularly interesting. In the section on diseases of the kidney the subjects discussed include glomerulonephritis and congestive heart failure and also the nephrotic hypertensive syndrome in diabetes and the intercapillary glomerulosclerosis of Kimmelstiel and Wilson.

The section on diseases of the heart and blood vessels is edited by W. D. Stroud. It covers a great deal of ground and occupies more than 140 pages. Even to mention all the main sections in this part is impossible in the available space. The diseases of the heart and blood vessels are dealt with in sections according to nomenclature revised by the Criteria Committee of the New York Heart Association. Special mention must be made of reports of cures in cases of bacterial endocarditis as a result of penicillin and heparin therapy, and of discussions on psychoneurosis and rheumatic fever. The section on anatomical diagnosis includes reference to thrombosis of the coronary arteries, and that on physiological diagnosis reference to the anginal syndrome. There are also sections on electrocardiography, treatment, and the peripheral blood vessels, as well as one devoted to miscellaneous subjects.

The last section on diseases of the digestive system and metabolism is edited by G. B. Eusterman. In the section on diseases of the œsophagus, stomach and duodenum peptic ulcer and its treatment are dealt with, particular reference being made to ulcer in service personnel. Two unusual subjects are noted—one on the treatment of peptic ulcer with the aid of posterior pituitary extract, and the other on peptic ulcer as an endocrine disease. In the section on diseases of the biliary tract and pancreatic system several references are made to hepatitis, and in the section on the intestinal tract a clinical and ætiological classification of diarrhoeal diseases is given. A section on diseases of metabolism and nutrition concludes the work.

This volume is comprehensive and valuable. The editors have made good use of the material available to them—almost entirely American and British; only one or two European journals are mentioned. The publishers deserve a word of praise for the quality of the printing and the general get-up of the work. They have had to conform to certain wartime requirements, and it is to their credit that the photomicrographs published are clear and leave little to be desired. The index is adequate.

¹ "Hypertension: A Manual for Patients with High Blood Pressure", by Irvine H. Page, A.B., M.D.; Fourth Printing; 1944. Springfield: Charles C. Thomas. 7 1/2" x 5", pp. 92, with seven illustrations. Price: \$1.50, post paid.

² "The 1944 Year Book of General Medicine", edited by George F. Dick, M.D., J. Burns Amberson, M.D., S.D., F.R.C.P. (Edinburgh and London), George R. Minot, M.D., S.D., F.R.C.P., William B. Castle, M.D., S.M., William D. Stroud, M.D., George B. Eusterman, M.D.; 1944. Chicago: The Year Book Publishers. 7" x 4 1/2", pp. 768, with 164 illustrations. Price: \$3.00, post paid. Australian price: 23s. 6d.

The Medical Journal of Australia

SATURDAY, MARCH 31, 1945.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

OPHTHALMOLOGICAL RESEARCH: HOPES FOR THE FUTURE AND A TASK FOR THE PRESENT.

MEDICAL RESEARCH has, as its ultimate object the prevention of disease and the discovery of measures that will promote the health and increase the general well-being of humanity. With this statement in mind it would not be difficult to name the objectives of research in ophthalmology. But if blindness is to be prevented and sight is to be preserved from deterioration, the magnitude of the task must be realized, and those who are qualified to undertake it or some part of it must see where their opportunity lies. Ophthalmology has a long history behind its present-day achievements. English ophthalmological history is said to have started with the Roman occupation of Britain. R. R. James in an early *British Medical Bulletin*¹ has traced the development of ophthalmology in Britain and has shown that although the foundations of modern ophthalmology were laid in the eighteenth century, it was not until early in the nineteenth century that rational ophthalmology displaced charlatanism. The Ophthalmological Society of the United Kingdom was founded in 1881 and its *Transactions* have been published regularly ever since. Early in its history the Ophthalmological Society of the United Kingdom may be said to have laid the foundations of preventive ophthalmology when it drew attention to the ravages of *ophthalmia neonatorum*, showed that the condition was preventable and suggested certain measures directed to that end. That no action was taken until many years had passed was unfortunate, but this early attempt to reduce the incidence of *ophthalmia neonatorum* originated with ophthalmologists. No better illustration could be chosen to show that in preventive medicine certain labels, deliberately chosen or of fortuitous origin, become attached to clinical conditions. These do not always indicate the sphere of investigation needed or the type of worker who should be

engaged on it. In research, too, what we may call the proprietorship is not always clear. When a condition affecting a special organ or system of organs is investigated, those conducting the research may or may not be specially trained clinicians in that subject. If the research is a clinical one, then clearly special clinical experience is indispensable, but in a non-clinical matter experience in research is the *sine qua non*. In either case, especially in the clinical subject, the help of other workers is often essential and they are something like scouts bringing information to headquarters.

To obtain some idea of the magnitude of the problem with which ophthalmological research in Australia has to deal is no easy matter. Professor Arnold Sorsby, writing in the same *British Medical Bulletin* as R. R. James, estimates that the rate of blindness in Great Britain is 2.5 per thousand of population, and his criterion of blindness is: "so blind as to be unable to perform any work for which eyesight is essential." Though some difference of opinion may be expressed regarding Sorsby's criterion of blindness, there is no need to discuss the point at present. In his first presidential address to the inaugural meeting of the Ophthalmological Society of Australia (British Medical Association), Sir James Barrett stated that at the 1933 census there were 3,898 totally blind persons in Australia. No information was available regarding partial sightedness. Further, information is entirely lacking concerning the amount of progressive deterioration of eyesight that occurs in the community as a result of untoward conditions in industry or in ordinary everyday life. What is quite certain is that there is plenty of scope for research in the ophthalmic field. Memories may be refreshed by an enumeration of the conditions mentioned by Sorsby in his discussion on the prevention of blindness. First come infective processes. *Ophthalmia neonatorum* has been all but abolished as a cause of gross visual defect and blindness, and such conditions as smallpox are no longer a significant cause of eye disease. It is the acute ophthalmias, tuberculosis and syphilitic lesions of the eye and trachoma which exemplify the problems of ocular disease. Lately rubella, a condition not mentioned by Sorsby (he was writing in 1943), has assumed some significance as a cause of cataract in infants, and further reference will be made to this later on. Sorsby then mentions genetic affections. He states that nowadays some 50% of blind children are blind from malformations and hereditary anomalies. He thinks that the present tendency not to make a rigid distinction between genetic and environmental factors is hopeful. "Genetic disease is no longer regarded as a fixed entity." It is known that environmental factors can excite or mask an inherent genetic tendency and Sorsby holds that though the basis for *retinitis pigmentosa* and other abiotrophies is undoubtedly chromosomal, the mechanism whereby they become manifest may well be somatic. Sorsby's third heading includes nutritional and metabolic disorders. He states that an understanding of diabetic retinitis will probably not be reached from study of the fixed retina under the microscope or from the biochemical analysis of the blood. "Suitable methods for investigating the respiration and total metabolic activity of the intact eye will no doubt be evolved." In regard to his fourth group, cataract and glaucoma, Sorsby points out that the problem,

¹ *British Medical Bulletin*, Number 9, Volume 1, 1943.

as in metabolic disorders, is the evolving of methods of study of the normal and abnormal physiology of the eye. In England and North America, he states, cataract and glaucoma are responsible for 25% to 40% of all blindness. His last group is that of injuries and we are reminded once again that war is the greatest single cause of blindness.

Sorsby discusses the organization of ophthalmology in the future. He is, by the way, the first holder of the research professorship in ophthalmology which was established in 1943 jointly by the Royal College of Surgeons of England and the Royal Eye Hospital, London. He holds that the future of ophthalmology, as of all clinical studies, depends on constant invigoration from new investigations dealing with changed conditions and expanding older knowledge. He continues: "Ophthalmological research as a career is an essential need in the reorganization of ophthalmic practice that is proceeding today. In England there is the prospect of creating a well-organized national eye service which will bring the benefit of specialized knowledge, under satisfactory conditions, to the whole population. In such a reorganized eye service, ample provision should be made for ophthalmic research institutes, where problems can be studied away from the pressure of the clinic, and where ophthalmic needs can be visualized from a better vantage point than the crowded out-patients' department." It should be pointed out that since those words were written a professorship of ophthalmology has been created at Oxford for purposes of research and treatment and that the first occupant of the chair is Miss Ida Mann, who is well known to Australian ophthalmologists. This is all of great interest to Australian medicine; and Australian doctors, and particularly the ophthalmologists among them, will wish to see Australia following the example set at Oxford. Such an event is not for the immediate future. What must be done now is work that lies at hand, and an important example can be given. On previous occasions attention has been drawn in this journal to the observations of Gregg in 1943 on the occurrence of cataract in the eyes of newborn babies whose mothers have suffered in the early days of their pregnancy from an attack of rubella, and to the extension of this work by Swan and others in Adelaide. In this issue we publish another communication by Gregg and a paper by D. G. Carruthers in which the observations include deaf mutism occurring among children whose mothers have a history similar to those reported by Gregg. These clinical observations, which reflect the greatest credit on those who made them, open up possibilities for future research that may be very far-reaching. This will be a task for the laboratory worker—physiologist, embryologist, bacteriologist and among others possibly the biochemist. Gregg in his paper refers to views of Professor Ida Mann which bear on this subject. But the clinical research is not ended. Gregg mentions some steps that are being taken, and it is clear that Australian ophthalmologists still have the task of making detailed observations on fresh cases of congenital cataract that occur in their practices, even as otologists have in regard to cases of deaf mutism. The important suggestion has also been made that detailed observations should be made in cases in which mothers who suffer from rubella during pregnancy do not give birth to infants suffering from a congenital

abnormality. Clearly there will also be something to do for the general practitioner, the "scouts" who should bring the information to "headquarters".

Current Comment.

MEASLES.

It often seems a pity that opportunities for presenting clinical studies of common epidemic diseases are lost because of lack of time, and perhaps lack of sustained observation by over-worked practitioners. It is perhaps felt that such subjects are trite. Nothing could be less true. Measles is an excellent case in point. It is almost universal, its morbidity is not insignificant and neither is its mortality from its complications, and it presents two features common in epidemic diseases. These features are variations in its signs and symptoms from epidemic to epidemic, and a steady alteration in its sweep and power. R. E. Smith, Medical Officer of Rugby School, in a review of the disease, claims our interest at once.¹ He remarks that the usual text-book description does not accurately apply to measles in children whose nutrition, housing and general environment are of high grade. This observation is based on his study of measles in a community of boys of well-to-do parents, living a well-ordered, careful, active life. He revives again interest in Goodall's description made only twenty years ago of the so-called illness of infection. This phenomenon is the almost immediate appearance of pyrexia during the first day or two of the incubation period, with evanescent symptoms suspicious of measles which subside until the usual incubation period is complete and then reappear in their classical declared form. He agrees with other observers that it is seen in 10% of cases if sought for. Prodromal rashes he has found exceedingly rare. When recognizable signs appear the rash is naturally one of the most important of these. Smith considers the enanthem is more valuable in diagnosis than the exanthem, since it appears earlier and is equally characteristic. The soft palate is studded with small elevations round which stellate patterns of tiny blood vessels are radially disposed. Influenza and streptococcal and pneumococcal infections may also cause such an appearance. A day or two later the Köplik's spots appear. Smith has found that these commonly spread from their original site inside the cheek near the second molar tooth to a much wider distribution over the mucosa of the cheeks and even to the gums. In 78 cases carefully charted he found that these spots appeared before the rash 21 times, on the same day as the rash 23 times and after the rash 12 times. In thirty cases they were not seen at all. Smith thinks they may be due to tiny vascular thrombi in the engorged vessels seen earlier. He has also seen the spots described by Hermann as appearing on the tonsils about the same time as Köplik's spots; but only about one-quarter of the boys had tonsils.

The question of the frequency of coryza in measles is interesting. Amongst the Rugby schoolboys coryza has been uncommon, in spite of the frequency of enanthems in nose, mouth and pharynx. It is suggested that the common coryza described in the classical accounts of measles represents rather the condition seen among poorer children. The exanthem conformed to the description of Sydenham in the seventeenth century.

Since no specific treatment of measles is known except serum therapy, studies in immunity assume a particular importance, even though the disease, like scarlet fever, is apparently becoming less virulent. The so-called "latent" immunity referred to by some writers is really, as Smith maintains, temporary immunity, which may break down later on further exposure to infection. Subclinical attacks are unproven; but whatever the explanation, it is true that certain individuals may resist infection, even when the degree of exposure is high, only to fall victim later.

¹ *Guy's Hospital Reports*, Volume XCIII, Numbers 1 and 2, 1944.

That there is an inherited immunity seems clear, as racial differences show. Also acquired immunity is for all practical purposes permanent. Of 513 boys leaving Rugby School over a period of five years, 493 had measles, but only six had second attacks, a small proportion even if it be supposed that no diagnostic errors were made.

One interesting point is whether an epidemic of measles becomes more virulent as successive waves of infection occur. In one epidemic specially studied at Rugby the disease became milder as it progressed.

The suppurative respiratory complications, due to secondary invaders, were found to occur more frequently only when the total numbers attacked were higher. It is significant to read that the incidence of complications was lowered by keeping the boys isolated in single-room "wardlets" until the crisis of fever was over. It is not altogether surprising to learn that these measures, together with the masking of all attendants, did not entirely prevent complications.

The literature on the subject of serum therapy is summarized by Smith. From this he considers that a dose of ten cubic centimetres of convalescent serum is effective as a preventive regardless of age, and a quarter to half of this dose will often cause attenuation. Adult serum, even if stored for several months or years, appears to be as effective as the serum derived from a person recently recovered from measles. To be effective it must be given early in the incubation period.

Smith attained some success in one epidemic by the use of filtered serum from volunteers. He has no doubt that the complication rate was quite misleading in assessing the value of serum. He believes that passive immunity will last at least only one epidemic season, but hopes that attenuated attacks may establish full immunity.

In conformity with general opinion the isolation of contacts has been given up at Rugby. It is, of course, admitted that children exposed to measles infection may prove a hazard to younger siblings, and it is in these cases that serum has a particular value.

Smith refers to the recent work on active immunization, and hopes that by using the virus cultivated on the chorio-allantois a safe and effective method of controlling measles will be worked out. In spite of the malign possibilities of this disease in very young children there is always the risk of the familiarity that breeds contempt; but the modern students of epidemiology and immunity are themselves an active antidote against such an attitude.

BLOOD CHANGES IN PRIMARY ATYPICAL PNEUMONIA.

PRIMARY atypical pneumonia, sometimes called "virus pneumonia", a name which involves an assumption, has been interesting the medical world for some time. Recent references to it in these pages have concerned chiefly its clinical features. It is a curious condition. At times its radiographic appearance mimics that of tuberculosis; it resists the therapeutic action of sulphonamides, and rarely it is associated with an encephalitis which may prove fatal. Of less practical interest are some of the blood changes; but these may yet prove to afford further insight into the nature and cause of the disease. Reports on the leucocyte counts carried out on patients with atypical pneumonia have not been very full or consistent up to date, so Ovid O. Meyer and Ethel W. Thewlis have set about supplying further information on the subject.¹ In a series of fifty cases they found the total white cell count to be substantially unchanged. The neutrophile cells and monocytes were somewhat increased in numbers, a finding which has been reported by others. There was usually a lymphocytopenia. The proportion of the eosinophile cells in all lung disease is a matter of interest, especially in view of cases of the type described by Löfner as "eosinophilic pneumonia" and the recently reported acarine infections of the lungs. Meyer and Thewlis found no increase in the eosinophile cells at the onset of the disease;

but they found some degree of eosinophilia in one-quarter of the late cases. Thus, a blood count is of a certain negative value in diagnosis, and may help to distinguish the disease from other forms of bronchopneumonia, though no positive findings are obtained.

More obscure is the strange finding that cold hæmagglutinins exist in the blood in atypical pneumonia. The American Commission on Acute Respiratory Diseases has published some work on this subject.² The earlier work on autoagglutinins which were only active in the cold was thought to indicate that some innate peculiarity, enhanced by several known pathological states, existed in the sera. Recently, however, it would appear to be not an infrequent finding in certain types of respiratory disease. It may be in some way related to these infections.

In all 214 cases were studied, most of these being instances of atypical pneumonia or pharyngeal or tonsillar infections, with a small number of other infections of the respiratory tract. A modified method of performing the test is described. The commission reports that higher titres of cold agglutination have been found in atypical pneumonia than in other types of respiratory infection. This is subject to some qualification, for the titre was also found to be proportional to the severity of the illness, as measured by the number of days of fever and the extent of the pulmonary lesion. Titres were found to rise during early convalescence and to fall thereafter. A strong titre thus is evidence in favour of a diagnosis of atypical pneumonia. This knowledge may be of value in the investigation of obscure cases where the laboratory staffs are sufficient to cope with the burden of yet another test. Nothing further is known concerning the mechanism and biological significance of this remarkable phenomenon. Like the Paul Bunnell test and the presence of some of the apparently meaningless agglutinins in normal blood, there are the elements of mystery in it. Perhaps some day some broader generalizations may be possible; but in the meantime it is to be hoped that further research will go on with no less zeal because the immediate goal is obscure.

BIOPSY OF THE THYROID.

IN the investigation of thyroid disease histological examination of aspirated thyroid tissue has been employed by Raymond E. Lipton and Martin S. Abel.² They give a careful description of the method they employ. They have found that if it is explained to the patient, the procedure is accepted as casually as the test for estimation of the basal metabolic rate. In a series of 23 cases they "had no untoward results from the procedure, nor any unpleasantness of any kind". In the first seven cases aspiration was performed immediately prior to thyroidectomy. In only one of these was there any evidence of injury to the gland. The aspirated tissue is fixed in formation and embedded in paraffin. Sections are cut in the ordinary way. The diagnosis depends on the size of the acinar cells, which become enlarged in thyrotoxicosis. According to Abel the average height (from base to free surface) of an acinar cell in healthy thyroid tissue should be less than 7.0 μ . In toxic nodular goitre it is 8.1 μ or more, and in toxic diffuse goitre it is more than 7.0 μ . Lipton and Abel failed to obtain sufficient material for examination in only three of 23 cases. Thyroidectomy was done in 17 cases. In all but one of these the biopsy findings agreed with the pathological diagnosis. The exceptional patient was a diabetic and a narcotic addict; his acinar cells were found to be no higher than would be expected in a nodular goitre, yet he was found to have thyrotoxicosis. There may be a place for aspiration biopsy of the thyroid in suspected thyrotoxicosis; but medical practitioners should be counselled to approach the task with caution. For the present it should be undertaken only when the surgeon is ready to proceed with operation.

¹ *The American Journal of the Medical Sciences*, December, 1944.

² *The American Journal of the Medical Sciences*, December, 1944.

¹ *Annals of Internal Medicine*, December, 1944.

Abstracts from Medical Literature.

PHYSIOLOGY.

The Oscillometer and Thermocouple as Diagnostic Aids in Peripheral Vascular Disease.

C. MOSES AND M. B. FERDERBER (*The Journal of Laboratory and Clinical Medicine*, November, 1944) describe an attempt to correlate the evidence obtained by oscillometer and thermocouple with the circulation in the extremities examined. Observations relative to the peripheral circulation were made on 102 individuals. This group included 48 normal subjects, eight asymptomatic, six with *thromboangiitis obliterans*, and 28 with arteriosclerosis. Observations as to history, symptoms, physical signs, oscillometric readings, skin temperatures, blood flow, and vibratory sensation, were noted and the results tabulated. The techniques used in obtaining the data are briefly described. While in arteriosclerosis of the lower extremity symptoms referable to the calf were most common, pain was noted almost as frequently in the ankle, arch, dorsum or toes. Numbness, tingling, burning, aching and rest pain were symptoms noted almost as frequently as intermittent claudication in arteriosclerotic vascular disease. A feeling of local fatigue was often the first sign of vascular disease. Arteriosclerotic rest pain was often relieved by moderate exercise. The pain of *thromboangiitis obliterans* was not relieved by exercise in any of the patients. Normal distribution of the hair over the lower extremity was noted in only 25% of the patients with vascular disease. Rubor or cyanosis or pallor of an extremity may be present in the absence of arterial vascular disease. The absence of the *dorsalis pedis* or posterior tibial pulsation was not found to be pathognomonic of vascular disease, and the presence of a pulsation did not exclude vascular disease. The oscillometric readings were not necessarily found to be decreased in arteriosclerotic peripheral vascular disease; normal persons with heavy musculature may have diminished oscillometric readings. Persons with vascular disease may have normal skin temperature. Estimation of the blood flow by Stewart's calorimetric method indicated that the blood flow in patients with vascular disease averaged about two-thirds of that in normal subjects. The authors found that estimation of the vibratory sense by the method of Barach yielded confirmatory evidence of deficient circulation, but was of little early diagnostic value. The arteriovenous anastomoses are suggested as one mechanism partially explaining the variations in skin temperature that occur in normal persons and those with peripheral vascular disease.

Changes in Phosphate of Muscle during Tourniquet Shock.

J. L. BOLLMAN AND E. V. FLOCK (*The American Journal of Physiology*, September, 1944) describe the changes in muscle following tourniquet shock. They state that many reports on shock indicate that the absorption of products of tissue autolysis from the area of

injury may be an important factor in the development of shock. While no toxic substance has been isolated and identified from the blood of man or animals in shock, there are many experiments which indicate the possibility of the production of shock by blood carrying toxic substances from regions of tissue autolysis. They draw attention to Green's work. Green made an extensive study of shock produced after the release of tourniquets which occluded the flow of blood to the hind limbs of animals for several hours. He found that shock was not produced unless occlusion was maintained for more than three hours before release and that intravenous administration of plasma did not prevent shock, nor did repeated administration of plasma prevent death. Shock was prevented by amputation or tight binding of the limbs shortly after the release of the tourniquets. Animals recovered from shorter periods of application of tourniquets and many short periods with recovery periods could be instituted without lethal effect. Green prepared extracts of normal muscle which were toxic on intravenous injection and produced a shock syndrome that terminated fatally on intramuscular injection. The authors' results indicate that the changes which occur in the phosphates of muscle, the blood supply of which has been completely occluded, are those of autolysing muscle. Adenosine triphosphate almost disappears after three hours and phosphocreatine is almost completely hydrolyzed in one hour. The inorganic phosphates of the muscle rapidly increase to a maximum in about four hours. The total of the acid-soluble phosphates is not changed. If the flow of blood is restored to the muscle within three hours there is resynthesis of adenosine triphosphate and phosphocreatine with a corresponding decrease of the inorganic phosphate. Fatal shock does not develop even though large amounts of muscle have been occluded. When the occlusion is released after more than three hours there is no regeneration of adenosine triphosphate or phosphocreatine, but considerable inorganic phosphate is washed from the injured muscle into the blood. Fatal shock develops in rats so treated if the muscles of more than one leg and thigh have been occluded for three and a half hours or if the occlusion of only one thigh and leg has persisted for six hours before release. This type of shock is not due to adenosine triphosphate washed out of the muscle, because adenosine triphosphate is destroyed during the occlusion and its decomposition products appear to be relatively non-toxic. In rats surviving release after occlusion of one leg for four hours, almost complete necrosis of the injured muscle occurs; but sufficient cells remain alive to restore function. After four to six weeks restoration of function of the leg occurs, although the original size of the muscle bundles is not completely restored.

The Effect of Anoxic and Anæmic Anoxia on the Motility of the Small Intestine.

E. J. VAN LIEKE, D. W. NORTHUP AND J. C. STICKNEY (*The American Journal of Physiology*, November, 1944) report some experiments concerning the combined effects of an epinephrine potentiating agent (cocaine) and anoxic

anoxia and of cocaine and anæmic anoxia on the propulsive motility of the small intestine of the dog. It had been established previously that neither anoxic anoxia nor cocaine alone had any appreciable effect on the motility of the small intestine. It had been shown, however, that hemorrhage equal to 3% of the body weight caused a significant acceleration of the motility of the small intestine. It was observed that if the animals were given cocaine and then subjected to anoxic anoxia, a statistically significant decrease in the propulsive motility of the small intestine occurred. Further, if cocaine was given to an animal which had suffered a hemorrhage equal to 3% of the body weight, anæmic anoxia no longer produced an acceleration but rather a statistically significant decrease in propulsive motility. The interpretation of these findings is that in the first instance cocaine produced a synergistic action with anoxic anoxia; the sympathetic nerves were stimulated and the motility of the small intestine was decreased. In the second instance, that is, following hemorrhage, cocaine again produced a synergistic action with the sympathetic nerves, so that during anæmic anoxia the action of these nerves predominated over that of the parasympathetics. When animals were subjected to combined anoxic anoxia and anæmic anoxia (without the influence of cocaine), the anoxic anoxia, by stimulating the sympathetic fibres, prevented the acceleration of the propulsive motility of the small intestine ordinarily produced by anæmic anoxia alone.

Mitotic Activity of the Corneal Epithelium.

J. S. FRIEDENWALD AND W. BUSCHKE (*The American Journal of Physiology*, July, 1944) report the effect of excitement and some drugs on the rate of mitosis in the corneal epithelium of rats. Excitement or annoyance of rats, particularly by painful stimuli, diminishes the mitotic rate in their corneal epithelium. The decrease of mitotic rate caused by excitement is an adrenergic response and can be simulated by local or systemic application of epinephrine. Both ergotamine and nicotine diminish the inhibition of mitosis following excitement. The effect of epinephrine is not due to a decrease in tissue temperature or to local circulatory disturbances. Removal of the superior cervical ganglion leads to a decreased mitotic rate in the rats' corneal epithelium after a period of about twenty hours. The authors suggest that this effect is attributable to the absence of some factor, other than sympathin, which regulates mitotic activity in this tissue under physiological conditions, and that this manifestation of denervation may be related to that of sensitization.

Energy Expenditure in Swimming.

P. V. KARPOVITCH AND N. MILLMAN (*The American Journal of Physiology*, August, 1944) report the results of investigations to determine the expenditure of energy in the various types of strokes used in swimming. The energy cost of swimming the crawl stroke, back stroke, breast stroke, butterfly stroke and side stroke at speeds greater than two feet per second was estimated. Twenty-four subjects of both sexes were observed. Swimming

at speeds higher than two feet per second should be considered a vigorous exercise because metabolism rises to more than ten times the basal rate. At speeds beyond five feet per second metabolism may be more than 100 times higher than basal. Unskilled swimmers expend from two to five times the energy used by skilled swimmers. Fluctuation between individuals is greater for the lower speeds than for the higher ones. The swimming strokes may be arranged in order of increasing energy cost as follows: crawl, back, breast and side. This relationship holds true for any corresponding speed. The butterfly stroke, however, has certain peculiarities. It is the least economical of the five strokes under 2.5 feet per second. Above this speed it becomes more efficient than the side stroke, and at three feet per second it is more economical than the breast stroke. A greater fatiguing effect of the butterfly stroke as compared with the breast style may be due to local fatigue of the shoulder girdle muscles. No sex difference in the relative energy cost of swimming was observed.

BIOCHEMISTRY.

Hypertension.

R. F. HOLDEN (*The Journal of Clinical Investigation*, November, 1944) records the results of studies on the reducing and nitrogenous fractions in protein-free blood filtrates. Such filtrates from subjects with and without arterial hypertension have been examined for non-fermentable reducing substance and non-urea nitrogen content. Appreciable amounts of non-fermentable reducing substance were noted in 30 out of 38 zinc filtrates from patients with hypertension disease and in four out of 21 control filtrates. A similar difference was found in tungstic acid filtrates. Among the hypertensive subjects, the non-preventable reducing substance in zinc filtrates exhibited some tendency to vary with non-urea nitrogen. No direct relation to blood pressure or urea clearance could be demonstrated.

Plasma Proteins.

W. COX AND A. J. MUELLER (*The Journal of Clinical Investigation*, November, 1944) have investigated serum albumin regeneration as affected by intravenously and orally administered protein hydrolysates. Enzymic hydrolysates of casein, lactalbumin and beef serum protein, which are nutritively equivalent to the original proteins, are equally effective in the regeneration of plasma albumin in hypoproteinaemic dogs, whether given orally or intravenously.

Thiouracil.

R. H. WILLIAMS *et alii* (*The Journal of Clinical Investigation*, September, 1944) have investigated the absorption, distribution and excretion of thiouracil. It is very rapidly absorbed from the gastro-intestinal tract and is readily excreted in the urine. When it was given in a dose ranging from 0.2 to 1.2 grammes daily, the concentration of the substance in the blood was found to vary from 0.8 to 6.4 milligrammes per 100 cubic centimetres, while the daily excretion in the urine varied from

16.0 to 618.0 milligrammes. Most of the thiouracil in the blood is in the cells, the red cells containing a larger total amount but smaller concentration than the white cells. Patients receiving thiouracil for several days preceding death were found at autopsy to have some of the substance in essentially all of the tissues of the body. Thiouracil was sometimes found in very large quantities in the bone marrow, thyroid, ovaries and pituitary, while striated muscle, testes and liver possessed relatively small concentrations. Adenoma of the thyroid gland possessed a much greater concentration of thiouracil than did relatively normal thyroid tissue. Cerebro-spinal, edema and pericardial fluids were found to contain distinctly less thiouracil than did whole blood; the concentration in pleural and ascitic fluid was about equal to that of blood, whereas milk contained about three times as much. Thiouracil is rapidly destroyed by the contents of the stomach and the small intestines. It is also rapidly destroyed by many tissues of the body. No thiouracil is excreted in the stools. The colon bacillus does not account for its absence, as it does not destroy the substance.

Iron Absorption.

THE absorption of ferrous and ferric radioactive iron by human subjects and by dogs has been studied by D. V. MOORE *et alii* (*The Journal of Clinical Investigation*, September, 1944). Comparison was made in the same normal and iron-deficient human subjects and in the same normal and iron-deficient dogs of the degree to which comparable test doses of ferrous and ferric radioactive iron were assimilated. The quantity of iron given varied from 1.0 to 4.0 milligrammes of iron per kilogram of body weight, and it was given under fasting conditions. The amount of radioactive iron which subsequently appeared as haemoglobin in the peripheral blood was used as the measure of the amount absorbed. Under these conditions, human subjects absorbed one and a half to fifteen times more ferrous than ferric iron, while dogs either absorbed both valency forms to a comparable degree or showed preferential assimilation of ferrous salts. Because different species of animals have been shown to differ in this respect, discussion of the valency form in which iron is most readily absorbed from the intestinal tract should be related to the species under consideration.

Water Deprivation.

A. W. WINKLER *et alii* (*The Journal of Clinical Investigation*, September, 1944) have studied balances of water, nitrogen and electrolytes during water deprivation, starvation and the ingestion of various solutions and foodstuffs. Fasting increased the negative water balance during water deprivation. The loss of fluids and electrolytes occurred at first predominantly from the extracellular phase and subsequently from the intracellular. No clear advantage of hypotonic saline solution over fresh water could be demonstrated in the amelioration of dehydration. Carbohydrate decreased the negative nitrogen balance, the ketone formation, the urinary volume, and the dehydration of completely deprived subjects. Its water of oxida-

tion was also made available to the body. All of the water of ingested fish was used to excrete the protein metabolites and therefore failed to minimize dehydration. The authors remark that under conditions of limited water supply, ingestion of protein foods is contraindicated. Carbohydrate is the foodstuff of choice.

Thiourea.

L. C. CHESLEY (*The Journal of Clinical Investigation*, September, 1944) has made observations on the absorption, apparent volume of distribution, and excretion of thiourea. Thiourea is not suitable for measuring total body water. When doses of 1,000 milligrammes are given by mouth, about 25% of the ingested thiourea cannot be recovered. None of the ingested thiourea appears in the faeces obtained over a three-day period afterwards. The concentration of thiourea in breast milk approaches very closely the concentration in serum. Thiourea enters the cerebro-spinal fluid, probably slowly. The renal clearance of thiourea is very close to that of urea.

Protein Metabolism.

R. M. FINK *et alii* (*The Journal of Experimental Medicine*, December, 1944) have studied plasma protein metabolism, both the normal and that associated with shock. Labelled plasma proteins were produced by administering to dogs the amino acid lysine synthesized with heavy nitrogen. Such labelled proteins are apparently indistinguishable biologically from proteins of normal isotope concentration. Labelled plasma proteins, as plasma, injected into normal dogs passed out of the blood stream at an initially rapid but constantly decreasing non-logarithmic rate. This outflow was balanced by a simultaneous inflow of plasma proteins from the tissues. Fifty per centum of the labelled protein was out of the blood stream in about 24 hours; 75% in about six days. Shock due to trauma of intestine or leg showed a dilution curve of labelled plasma protein not unlike that of the normal dog. If anything, dilution appeared a little less rapidly in shock. Since the usual shrinkage of plasma volume and plasma protein mass was present in these shocked dogs, these data were compatible with a decreased inflow of protein into the plasma during shock. These data may indicate that the plasma proteins are normally in constant and rapid exchange with a mobile pool of body protein.

Riboflavin.

THE riboflavin content of the food served in Royal Air Force messes has been determined by T. F. MOORE *et alii* (*The Biochemical Journal*, Number 2, 1944). Biological and microbiological methods were used. The values obtained by the two methods were in good agreement, though the results obtained by the biological method were somewhat higher than those obtained by the microbiological method. The daily intake of riboflavin in these messes ranged from 1.5 to 2.6 milligrammes per person and averaged about 1.9 milligrammes. Since no sign of deficiency of riboflavin exists among the persons receiving these diets, it is considered that the average riboflavin requirement of adults does not exceed 2.0 milligrammes daily.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held on December 14, 1944, at the Robert H. Todd Assembly Hall, British Medical Association House, 135, Macquarie Street, Sydney, Dr. G. C. WILLCOCKS, the President, in the chair.

Congenital Defects following Maternal Rubella.

DR. N. MCA. GREGG read a paper entitled "Rubella during Pregnancy of the Mother, with its Sequelae of Congenital Defects in the Child" (see page 313).

DR. D. G. CARRUTHERS read a paper entitled "Congenital Deaf-Mutism as a Sequela of a Rubella-Like Maternal Infection" (see page 315).

DR. DONALD VICKERY expressed appreciation of the papers. He said that he had on record 21 children with congenital defects who had come to his care in 1943-1944; he proposed to describe the main features which the children presented. The ophthalmic surgeons examined the majority of babies with cataract in 1941-1942, and the association of cataract with congenital cardiac disease was well recognized; but it was not until the end of 1942 and the beginning of 1943, when these children reached the age of two years, that the pediatricians and the ear, nose and throat surgeons became aware of the syndrome in children who had no noticeable eye defects whatsoever. Dr. Vickery said that of his 21 patients, only two had congenital cataract. Most of the mothers had consulted him because the child presented one or more of certain symptoms; they were: (i) general backwardness (the child was not attempting to speak, or the mother thought he was deaf); (ii) failure to thrive; (iii) suspected heart trouble (the mother had perhaps been told that the child had a leaking valve); (iv) difficulty of management—the child was a problem child. All the 21 children were born between December, 1940, and August, 1941; 20 of the mothers gave a history of an attack of German measles during the first three months of pregnancy. Dr. Vickery said that the remaining child was so typical, with his deaf-mutism, failure to thrive, congenital cardiac lesion *et cetera*, that there was not the slightest doubt that the mother had suffered from German measles early in her pregnancy.

Dr. Vickery went on to say that all the children showed a variable degree of deaf-mutism; all were backward in speaking, and not one was as yet able to put sentences together. No child was totally deaf, but all were deaf to the extent that they did not yet understand and comprehend what was spoken to them. Most of them could hear loud, sharp sounds, such as a hand clap, a whistle or a telephone bell. Thirteen of the children had weighed less than six pounds at birth, though the majority of them were full-time babies. Eleven were grossly undersized and underweight; they weighed less than two stone at the age of three and a half years, and some weighed 21 and 22 pounds. Thirteen showed definite evidence of a congenital cardiac defect, with cardiac murmurs, precordial bulging and cardiac enlargement. Five of these had a machinery type of murmur over the pulmonary area, strongly suggestive of a patent *ductus arteriosus*. One of the five had been subjected to operation in July, 1944, by Dr. T. Y. Nelson, of the Royal Alexandra Hospital for Children; the *ductus arteriosus* had been successfully ligated, and the child's physique and development improved and the machinery murmur completely disappeared. The remaining eight children with cardiac lesions had mainly systolic murmurs over the precordium, with the greatest intensity over the third left intercostal space; their condition was strongly suggestive of a patent interventricular lesion. X-ray films of a number of these children chiefly showed left ventricular dilatation and suggested the presence of an interventricular lesion or aortic stenosis. Dr. Vickery said that four children with congenital cataract had been examined *post mortem* at the Royal Alexandra Hospital for Children by Dr. D. H. K. Reye; all had patent *ductus arteriosus*, and two had interventricular septal lesions as well.

Dr. Vickery went on to say that most of the 21 children had a general instability of the nervous system; they lay awake for hours at night (this was especially noticeable during the first two years of their life), they were unable to concentrate, and they displayed a peculiar fidgeting, prying interest in things. During the fourth year of life the power of concentration of many of the children had greatly improved, and many were learning to cooperate with their

hands and eyes, and in this way were developing their minds; the faculty of hearing and speech was still much retarded. The future outlook for those whose physical development had not been greatly retarded by their cardiac lesions was reasonably good, in view of the noticeable improvement in concentration that occurred, and it seemed that they would be able to be taught some useful trade through the cooperation of their eyes and hands. Dr. Vickery was sure that many would learn to speak. The problem of preventing a similar catastrophe in the future to another crop of children was no easy one; in order to prevent anxiety amongst pregnant women careful consideration and handling would be needed, especially in view of the multitude of conditions which produced a rash simulating that of German measles.

DR. A. J. GIBSON said that, as an obstetrician, he felt exceedingly guilty to think that so many cases of congenital cataract were first diagnosed by an ophthalmologist. Dr. Gregg had done a great service in stressing one aspect of the subject—the possibility that certain other infectious diseases during pregnancy might be followed by other congenital defects. Dr. Gibson said that ever since Dr. Gregg had published his facts, he (Dr. Gibson) had been doing his best to teach students to record positive findings during pregnancy, no matter how trivial. Dr. Gibson could not speak from personal knowledge, but he understood that during the epidemic of German measles there was quite a high rate of stillbirths or premature births. One general practitioner had told him that in his practice several children had been lost, and that at the hospital at which they were born the matron had reminded him that various mothers of stillborn or premature infants had suffered from German measles early in pregnancy. Dr. Gibson thought that that might be another factor to be taken into account. He had had three patients suffering from German measles, in one case complicated by severe toxemia; when that woman's baby was born he had with some difficulty unfortunately kept it alive. Later he had found that it had congenital cataract; subsequently it developed rather badly, and the outlook was poor. A second baby was deaf. Dr. Gibson said that he had been interested in Dr. Vickery's statement that in every one of his cases in which the mother had suffered from German measles early in pregnancy, the child had some congenital defect. That observation raised an important question: was one justified in allowing such a pregnancy to go on? Pregnant women themselves would be asking the same question. If it was found that German measles in early pregnancy was invariably followed by congenital defects in the baby, was it right to allow the pregnancy to continue? The problem required serious consideration. One of Dr. Gibson's patients had suffered from German measles in pregnancy, and there was great anxiety as to whether the baby would be born healthy or not; its eyes seemed normal and its heart appeared normal, but some defects might appear in the hearing or the teeth. That mother had had German measles at a later stage in pregnancy. In conclusion, Dr. Gibson thanked Dr. Gregg and Dr. Carruthers for their papers.

DR. G. C. HALLIDAY asked for information as to the latest stage in pregnancy at which German measles occurred and was followed by congenital defects in the infants. Dr. Halliday said that he had only six cases in his private practice, and one of the mothers had had rubella in the latter part of the fourth month of pregnancy. He wondered whether any of those present had had a similar occurrence.

DR. E. S. A. MEYERS also thanked Dr. Gregg and Dr. Carruthers. He said that both the papers were based on original work, and were thus of greater interest. In answer to Dr. Halliday's question, Dr. Meyers said that in the series of nearly 150 cases recorded at the Department of Public Health, no definite congenital defects had been recorded as following maternal rubella occurring after the fourth month; in a number of cases defects had occurred when the mother had suffered from rubella in the fourth month. Dr. Meyers thanked all those doctors who had notified the health department of cases in their experience; he said that it was not an easy matter to find time to do so when everyone was so busy, but the problem was viewed very seriously. The health department had 145 cases and Dr. Gregg had recorded 78; many no doubt had not yet been recorded, so that a total of more than 200 cases had occurred as a result of one epidemic. Dr. Meyers said that he was afraid that doctors were going to be asked to do even a little more; a circular was to be sent out to every doctor asking for particulars of cases that might occur in the future. Information concerning "negative" cases had not so far been sought. Dr. Meyers said that he had heard from one woman who had suffered from rubella in the third

month of pregnancy and whose child seemed normal. Another woman who had had rubella in the third week of pregnancy had an apparently normal child. This last-mentioned case might upset the 100% of congenitally defective infants so far recorded when the infection occurred in the first two months of pregnancy. Dr. Meyers said that it was necessary to record "negative" cases. All maternity hospitals had been asked to question women entering hospital for their confinement. Dr. Meyers went on to discuss the question whether the condition was new. He thought that it was not new. He knew of one girl, aged twenty years, whose mother had suffered from rubella during the third month of pregnancy. The girl was a deaf mute, undersized and suffering from microcephaly; although her training had been much retarded, she was a clever commercial artist. Two cases had been reported in *The Lancet*, in which the children were born "during the past ten years". The condition was apparently not new. Dr. Meyers thought that every one of the children should be radiologically examined. Referring to the question asked by mothers, whether all their children would be similarly affected, Dr. Meyers said that the mothers could be reassured; in following pregnancies, in all cases recorded, the children were normal.

Dr. E. P. BLASHKI said that he had had the privilege of a ringside seat at the beginning of Dr. Gregg's investigation. Dr. Blashki thought that care was necessary in estimating the number of these deaf children who had been examined by otologists; many of them had been taken to more than one otologist, and so would be recorded several times over. Dr. Blashki said that he had been struck with the fact that the older children had some perception of sound. He had been interested to try to find what could be done for them and to console the parents a little and teach them to overcome the difficulty. Otologists had always been in the habit of saying to parents: "Yes, your child is deaf, but not dumb in either sense." But in the present cases care was necessary; it was not always possible to make such a statement, because the deaf child might prove to be subnormal and consequently difficult to educate. However, parents should be encouraged to try to educate these children. Dr. Blashki said that he had met with some parents who had received no encouragement at all from doctors. A great piece of work had been undertaken in the investigation of this syndrome, to try to prevent it from happening in the future; but time was going on, and all the affected children were reaching an age at which they were growing more difficult to train. The provisions in Australia for the training of deaf children were very inadequate. No more could be done until a great many competent teachers had been trained, and that process would take at least two years. Some of the children were now aged more than three and a half years; these children would not get the benefit of the trained teachers until they had reached normal school age. Dr. Blashki urged that the mothers should be taught to begin the children's education at home. In the literature a great deal was stated to the effect that only competent teachers should teach deaf children. But deaf children, like other children, did not spend all their time at school, and the mothers had plenty of opportunity to teach their children at home. It was difficult to find a way to teach the mothers. Dr. Blashki said that he had written to England for a pamphlet entitled "What the Mother of a Young Deaf Child Can Do"; he considered the pamphlet valuable enough to warrant wide distribution in Australia. There was also available in England a book entitled "All About the Deaf", which was a mine of information, and from America could be obtained "The Acoustic Method" and "The Problems of the Deaf", by Dr. Max Goldstein. Dr. Blashki urged that deaf children who had any hearing should be taught by sound, not by signs, so that they would develop into normal children, not finger-spellers. In conclusion, Dr. Blashki put forth a plea that something should be done at once, and that the matter should not be allowed to drop.

Dr. Gregg, in reply, referred first of all to Dr. Carruthers's question about the proportion of deafness amongst children thought to be suffering from purely ocular defects. Dr. Gregg said that of his last eight patients, three were known to be deaf, and in the last fortnight one other child had been found to be deaf. Dr. Gregg agreed with Dr. Carruthers that the deafness appeared much later and so was not recognized so early as the other defects; he thought that of the first group of 78 patients, probably some were deaf. Dr. Gregg again urged on otologists the necessity, when examining these deaf children, to look for ocular signs. He had been pleased to hear Dr. Vickery's statement that he had questioned the mothers of healthy children of this age

group as to the occurrence of rubella during pregnancy. Dr. Gregg said that when he read his first paper on this subject three years earlier in Victoria, the question arose of the possible incidence of the syndrome. One way of finding out was to question the mothers of healthy children of the relevant age group. During the whole period of the investigation, he found no mother of a healthy child who had suffered from rubella during pregnancy. Dr. Gregg was delighted to hear that Dr. Gibson was instructing his students to take histories carefully; Dr. Gregg would add that they must not only record any illness, but also the absence of illness. Otologists should watch for another possible crop of deaf mutes among the children born to mothers affected by the 1942 epidemic of rubella; if there were any deaf mutes among them, these should just about be coming to notice. Dr. Blashki had pointed out that some of the cases might have been doubly counted; Dr. Gregg said that that might be so in the records of individual otologists, but it was not true of the records of Dr. Meyers at the health department. Dr. Gregg was pleased to hear Dr. Blashki urge the need for education early and at home. He thought that one result of the meeting should be that the Section of Oto-Rhino-Laryngology of the Branch should make its presence felt as a body in demanding such education. The future of these children necessitated active education at once. Another aspect needed public attention. So far the welfare of the children had been the main consideration; attention should also be given to the effect produced on the other members of the homes to which these children belonged. In many of the severe ocular cases, the children were mentally deficient. There was no adequate provision for such children in New South Wales; only with difficulty could arrangements be made for their care after the age of three years. But by the time the child reached such an age, homes were disrupted. The mothers were asked to bear far too much. There should be some way of taking such a mentally defective child from the mother before she had to ruin her home and her life in looking after it.

Dr. Carruthers, in reply to Dr. Halliday, who had asked about the latest occurrence of rubella in the mother which had caused congenital defects in the infant, said that in the case of deaf mutes the organ of Corti seemed to be the part that was susceptible to virus damage, and this might occur even after it had developed. A similar process could be seen in children who suffered from mumps; sudden and severe damage to the inner ear could be caused. The effect might be the same as in the waltzing guinea-pig, in which degeneration and ultimate disappearance of the fully developed Corti's organ could be observed. In the case of five more deaf children, Dr. Carruthers had looked over their histories and found the occurrence of measles at the age of eighteen months, measles at fifteen months, meningitis at nine months, a "teething rash" (possibly scarlet fever) at eighteen months. The parents thought that these children became deaf after these infections. The organ apparently was susceptible even after it had developed. Dr. Blashki and Dr. Gregg had urged the early education of the children. Dr. Carruthers said that he had induced the parents to put microphones on their wireless sets and tried various means to get the children to realize that there was such a function as listening. Some parents had been persuaded to send the children to teachers, and the efforts made had brought about a great difference in the children. At the age of three or three and a half years progress had been tremendous. Dr. Carruthers said that he had had more than one child whose parents had been told to leave him alone until he was old enough to be sent to the special school for deaf mutes. Such an attitude was very wrong. Special training and education must commence from the very earliest age when interest could be stimulated.

Dr. Willcocks, from the chair, thanked Dr. Gregg and Dr. Carruthers for their clear presentation of an interesting subject. Dr. Willcocks also thanked the other speakers for their remarks, which had been exceedingly helpful. He thought it appropriate to say that Dr. Gregg had raised a hornet's nest; he seemed to have set at least half a dozen departments of medicine by the ears. All those present had learnt something, and Dr. Gregg had revealed to them an interesting series of events and pointed the way for further investigations in other fields of observation and for suggestions as to prevention and treatment. Dr. Willcocks hoped that either Dr. Gregg or somebody else would continue to stir up the hornet's nest and so widen the field of investigation and discovery. The field that had been opened up was remarkable, connoting as it did the possibility that congenital defects might follow other maternal infections than rubella during pregnancy. As in

so many other subjects that were brought up at similar meetings, there were many avenues for discussion, and it was difficult to know down which of them one should direct one's words; the speakers had gone down different lanes and given a good survey of the subject. Dr. Willcocks thanked Dr. Blashki for the books and pamphlets that he had brought to the meeting; he thought that they should be ordered for the Branch library. If the Section of Oto-Rhino-Laryngology would indicate the best way to make use of the publications, it would be done. Unless the Branch obtained some of the pamphlets, it was possible that nothing would be done.

NOTICE.

THE General Secretary of the Federal Council of the British Medical Association in Australia has announced that the following medical practitioners have been released from full-time duty with His Majesty's Forces and have resumed civil practice as from the dates mentioned:

Dr. A. W. McClaren, Kendal Street, Cowra (February 25, 1945).

Dr. G. H. Puddicombe, 27, Redman Parade, Belmore (March 12, 1945).

Post-Graduate Work.

COURSES AT MELBOURNE DURING 1945 FOR MEDICAL GRADUATES.

THE Melbourne Permanent Post-Graduate Committee announces that the following courses for medical graduates will be held during 1945. Particulars regarding fees, times and places may be obtained from the Secretary of the committee at the Royal Australasian College of Surgeons Building, Spring Street, Melbourne; telephone: Central 1855. Entries, accompanied by the appropriate fee, should be in the hands of the Secretary two weeks before the commencement of each course.

M.D. (Part II) and M.R.A.C.P.

A series of demonstrations suitable for candidates representing themselves for Part II of the examination for the degree of doctor of medicine, or for admission to membership of the Royal Australasian College of Physicians, will begin on April 5 and conclude on December 13, 1945.

M.S. (Part II) and F.R.A.C.S.

A series of demonstrations suitable for candidates representing themselves for Part II of the examination for the degree of master of surgery or for admission as fellows of the Royal Australasian College of Surgeons will begin on April 10 and conclude on December 11, 1945.

Refresher Course.

A refresher course of medical and surgical lectures and demonstrations, specially designed for general practitioners, will be conducted from June 25 to July 7, 1945. An endeavour will be made to provide residence for a number of those attending. Early notification of intention to attend will facilitate these arrangements. During the course a series of six lectures under the general title of "The Background of Infectious Diseases in Man" will be given by Dr. F. M. Burnet, Director of the Walter and Eliza Hall Institute of Research in Pathology and Medicine.

Diseases of the Chest.

A symposium on diseases of the chest will be conducted on five afternoons, from May 17 to June 14, 1945, by Dr. Hilary Roche, Dr. C. H. Fitts and Dr. C. J. O. Brown.

Malignant Tumours.

Dr. R. Kaye Scott will give a series of lectures on malignant tumours from August 6 to September 3, 1945.

Clinical Pathology.

A series of four lecture-demonstrations in clinical pathology will be given by Dr. Hilda Gardner from October 2 to October 23, 1945.

Neurological Clinico-Pathological Demonstrations.

Dr. E. Graeme Robertson will give a series of clinico-pathological demonstrations in neurology from September 21 to November 16, 1945.

University of Melbourne: M.D. (Part I) and M.S. (Part I).

Professor R. D. Wright, Professor P. MacCallum and Professor S. Sunderland, of the University of Melbourne, have arranged courses of instruction for the degree of doctor of medicine (Part I), which commenced on March 14, 1945. It is hoped to repeat these courses during the latter part of the year.

Correspondence.

"FOUND DEAD", "DEAD IN BED", AND "COLLAPSED AND DIED".

SIR: A case of topical interest following Professor Cleland's article of March 3, 1945, occurred here this week. A man of sixty-four years was found dead on his bed in a locked house on March 6, 1945. He had last been seen on March 3. I was asked by the local sergeant of police to see the body and advise him as to the nature of stains which he thought might be blood.

The hall of the house showed a well-defined track of footmarks in a thick smear of tarry faecal material. The body was found on a bed, clothed but for bare feet, which were fouled with melenæ. There were several patches of tarry stool on the floor and many footprints.

I had not seen the man before and no recent medical history was available. At autopsy the colon and ileum were loaded with tarry faecal matter. The stomach contained only a little dark blood-stained mucus. At the pylorus a deep peptic ulcer (diameter 0.75 centimetre) had eroded a large vessel. Death had followed gross hemorrhage.

The only medical record of this man available refers to an attack of retention of urine in 1941. Of recent years he is said to have drunk heavily, and I have no doubt that a bout preceded his death. Living on his own, he could not summon help, though the footprints suggest he may have tried.

Yours, etc.,
J. S. STOREY.

Zeehan,
Tasmania,
March 10, 1945.

INJURIES BY UNKNOWN AGENTS TO BATHERS IN NORTH QUEENSLAND.

SIR: We would like to reply to a letter from H. Flecker, of Cairns, which appeared in your journal of January 27, 1945.

It is pleasing to note that Dr. Flecker agrees generally with our statements that the Siphonophore, *Physalia*, was wrongly reported as the cause of many of the marine stings in Queensland. We are, however, still not clear on several points mentioned by your correspondent.

Dr. Flecker states that *Physalia* weals are unmistakable, and asserts that such weals are common amongst North Queensland bathers. This statement is challenged on the following grounds. To the knowledge of one of us (F. A. McNeill) there are no *Physalia* in North Queensland waters. This claim is backed by a wide experience over many years of the Barrier Reef area in different seasons of the year, and also after continuous inquiries directed to boating men and island residents. In view of this the presence of weals on bathers in the waters of northern Queensland must be attributable to an underwater and not to a surface floating form such as *Physalia*. We would like Dr. Flecker to prove to us that *Physalia* (Portuguese man-o'-war) does occur inside the Barrier Reef, so as to substantiate his identification of the causal organism in the two fatal cases he mentions—the ones in which post-mortem examinations were carried out. In our opinion there is another organism causing bad weals which have an excoriated appearance even after four to six days, and these are firmly believed to be those inflicted by the carybdeid medusa (jelly fish), known to frequent North Queensland waters. This medusa, however, is not necessarily the same species or even genus as the one responsible for killing the ten-year-old aboriginal boy at Darwin.

We make the suggestion that the weals produced by marine stinging animals from North Queensland be very critically examined for differences from characteristic *Physalia* weals, since the latter never persist for more than twenty-four hours or so, even in the very worst cases.

Since the writing of our article in *The Australian Journal of Science* (Volume V, Number 6, June, 1943) we have learnt of the numerous cases of mystery stings similar to those mentioned by Dr. Flecker, and where the lesion is not a weal. We agree with Dr. Flecker as to their dangerous nature.

Since medusæ are frequently so hard to distinguish below the surface of the water, we would suggest the use of fine tow-nets to catch stingers after victims have been brought ashore.

Yours, etc.,

F. A. McNEILL and ELIZABETH C. POPE,
Department of Invertebrates.

The Australian Museum,
Sydney,

March 15, 1945.

THE TREATMENT OF HÆMORRHOIDS BY INJECTION.

SIR: In the issue of March 10, 1945, of the journal, in reviewing a book by R. Rowden Foote, "Varicose Veins, Hæmorrhoids and other Conditions: Their Treatment by Injection", I note the following statement in connexion with the injection of hæmorrhoids: "whilst a solution of 20% phenol in glycerin, if used at all, should be used only by those expert . . . in these methods". I would like to point out that this solution is equally as safe as the more commonly employed phenol in almond oil.

I have used 20% phenol in glycerin for the past seven or eight years to inject internal hæmorrhoids in suitable cases. I do not claim to be an "expert in these methods"; but I have never experienced any complications providing the correct amount is employed, and I have found it more efficacious than phenol in almond oil. Even where there has been some leakage of the solution, no pain or sloughing has occurred. Of course, the solution must be injected into the pile mass itself, and not into the mucous membrane.

Yours, etc.,

H. BARNETT.

58, John Street,
Lidcombe,
March 10, 1945.

A DISCLAIMER.

SIR: I wish to disclaim responsibility for a report in *The Daily Telegraph* of March 22, 1945, of a talk on "A Nationalized Medical Service" which I gave before the Sydney University Medical Society.

Firstly, I did not know that any but medical students were present; and secondly, *The Daily Telegraph* gives an incorrect version of some of my statements.

I must in fairness add that after the discussion, while I was talking with a small group, a young man asked me if he might report some of the views expressed, but I did not realize that he represented *The Daily Telegraph*, taking it for granted that he was from the staff of a students' journal.

Yours, etc.,

E. P. DARK.

Katoomba,
New South Wales,
March 23, 1945.

BELL'S PALSY.

SIR: I would like to thank those doctors who have written to me on the subject of Bell's palsy and my contention that it is a virus disease closely connected with varicella and herpes zoster. One doctor had seen several cases in one day; another had seen Bell's palsy and herpes zoster concurrently on a patient.

The latest edition of Rose and Carless states that Bell's palsy is due to "inflammation from exposure to cold". My other text-books either agree with this aetiology or attribute it to "rheumatism". Major Ennis has, however, written to me pointing out that F. R. M. Walshe calls the condition "geniculate herpes".

In the large epidemic of varicella I witnessed last year it was no surprise when adults came along with herpes zoster, but I was surprised to see as many cases of Bell's palsy. Upon the termination of the epidemic of varicella I have seen no more cases of either herpes zoster or Bell's palsy.

I believe that these three conditions must be closely related, varicella affecting the young and herpes zoster and Bell's palsy the older. As Walshe calls Bell's palsy "geniculate herpes" and states that often the palsy is preceded by herpetic vesicles in the meatus, he must also think this condition due to a virus.

I am indebted to Miss M. Rolleston, librarian of the British Medical Association, Sydney, for Walshe's article.

Yours, etc.,

F. W. SIMPSON.

271, Cambridge Street,
Wembley Park,
Western Australia.
March 17, 1945.

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 54, of March 15, 1945.

PERMANENT NAVAL FORCES OF THE COMMONWEALTH (SEA-GOING FORCES).

Promotion.—Acting Surgeon Lieutenant-Commander Trevor Alexander McLean is promoted to the rank of Surgeon Lieutenant-Commander, dated 7th March, 1945.

ROYAL AUSTRALIAN AIR FORCE.

Citizen Air Force: Medical Branch.

Flight Lieutenant J. P. Gallagher (267409) is transferred from the Reserve with effect from 15th January, 1945.

Temporary Squadron Leader G. M. Oxer (251201) is granted the acting rank of Wing Commander whilst occupying a Wing Commander post with effect from 4th September, 1944.

The following temporary Flight Lieutenants are granted the acting rank of Squadron Leader whilst occupying Squadron Leader posts with effect from the dates indicated: W. A. Leventhal (263409), 1st November, 1944, T. J. Fennell (263864), 1st January, 1945.

The grant of the acting rank of Wing Commander to temporary Squadron Leader G. M. Oxer (251201) is terminated upon his ceasing to occupy a Wing Commander post with effect from 14th December, 1944.

Temporary Squadron Leader W. Deane-Butcher (261286) is granted the acting rank of Wing Commander whilst occupying a Wing Commander post with effect from 1st February, 1945.

The probationary appointments of the following Temporary Squadron Leaders are confirmed with effect from the dates indicated: N. A. Albiston (257550), 20th January, 1944, A. L. Webb (267631), 10th July, 1944.

The probationary appointment of Flight Lieutenant J. D. Whiteside (256812) is confirmed with effect from 1st April, 1945.

Reserve: Medical Branch.

The following medical practitioners are appointed to commissions on probation with the rank of Flight Lieutenant with effect from the dates indicated: Eric Bruce Lee (267793), William Michael Calanchini (257721), Frederick Joseph Scanlon (267791), Phillip Arnold Deck (267790), 1st January, 1945, Godfrey Chesworth Scott (267794), Chester Gilmore Wilson (277533), Edward Maxwell Diment (267792), 8th January, 1945, Edward Matthew Slattery (267795), 15th January, 1945.—(Ex. Min. No. 63—Approved 7th March, 1945.)

The following medical practitioners are appointed to commissions on probation with the rank of Flight Lieutenant with effect from the dates indicated: Albert Anthony Golden (267796), 1st January, 1945, Percy Norman Wood (277534), 8th January, 1945, Lynn David Walters (277535), 20th January, 1945.—(Ex. Min. No. 64—Approved 7th March, 1945.)

The following officers are transferred from the Active List with effect from the dates indicated: (Temporary Squadron

Leaders) R. Lloyd-Jones (261773), 5th January, 1945, G. M. Ozer (251201), 17th January, 1945.—(Ex. Min. No. 67—Approved 7th March, 1945.)

The following officers are transferred from the Active List with effect from the dates indicated: (Temporary Squadron Leaders) E. W. Field (251247), 12th January, 1945, J. F. Hughes (252290), 15th January, 1945, A. A. Murray (252893), 16th January, 1945.

Dr. Leonard Charles Doubleday, M.B., B.S. (267797) is appointed to a commission on probation with the rank of Flight Lieutenant with effect from the 1st February, 1945.—(Ex. Min. No. 77—Approved 14th March, 1945.)

CASUALTIES.

ACCORDING to the casualty list received on March 20, 1945, Major E. J. K. Harbison, A.A.M.C., North Adelaide, is reported to have died of illness.

Obituary.

SAMUEL JOSEPH HENRY MOREAU.

WE regret to announce the death of Dr. Samuel Joseph Henry Moreau, which occurred on March 20, 1945, at Sydney.

CEDRIC JAMES LOGAN.

WE regret to announce the death of Dr. Cedric James Logan, which occurred on March 20, 1945, at Inverell, New South Wales.

Nominations and Elections.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Walton, John William, M.B., B.S., 1943 (Univ. Sydney), 28, Jersey Street, Enfield.

Medical Appointments.

Dr. John Coffey, Deputy Director-General of Health and Medical Services, Queensland, has been appointed a member and chairman of the Queensland Radium Institute.

Dr. Harold George Rich has been appointed Government Medical Officer at Narrandera, New South Wales.

Dr. James Bruce Gordon has been appointed Visiting Medical Officer at Westwood Sanatorium, Queensland, in pursuance of the provisions of *The Charitable Institutions Management Act of 1885*.

Dr. Aylmer Edward Burke-Gaffney has been appointed Visiting Medical Officer at the Aboriginal Settlement, Woorabinda, Queensland, in pursuance of the provisions of *The Aborigines Preservation and Protection Act of 1933*.

Books Received.

"Stitt's Diagnosis, Prevention and Treatment of Tropical Diseases", by Richard P. Strong, M.D., Sc.D., D.S.M., C.B.; Seventh Edition, in two volumes; 1944. Philadelphia: The Blakiston Company. 9" x 6". Volume I, pp. 927, with many illustrations; Volume II, pp. 922, with many illustrations. Price: \$21.00 per set.

"Immuno-Catalysis", by M. G. Sevag, Ph.D., with a preface by Stuart Mudd, M.A., M.D., 1945. Springfield: Charles C. Thomas. 9" x 6". pp. 284. Price: \$4.50, post paid.

"Endocrinology of Woman", by E. C. Hamblen, B.S., M.D., F.A.C.S.; 1945. Springfield: Charles C. Thomas. 10" x 6½". pp. 277, with many illustrations. Price: \$8.00, post paid.

"The 1944 Year Book of Physical Medicine", edited by Richard Kovács, M.D.; 1944. Chicago: The Year Book Publishers, Incorporated. 7" x 4½". pp. 416, with 66 illustrations. Price: \$3.00, post paid; in Australia, 23s. 6d.

"Doctor's Conscience or All Illness is Preventable", by Stanley Boyd, M.R.C.S. (England), L.R.C.P. (London); 1944. Sydney: The Currawong Publishing Company. 7" x 5½", pp. 88.

"The 1944 Year Book of General Surgery", edited by Everts A. Graham, A.B., M.D.; 1944. Chicago: The Year Book Publishers, Incorporated. 7" x 4½", pp. 736, with many illustrations. Price: \$3.00, post paid; in Australia, 23s. 6d.

"Textbook of Gynecology", by Wilfred Shaw, M.A., M.D. (Cantab.), F.R.C.S. (England), F.R.C.O.G.; Fourth Edition; 1945. London: J. and A. Churchill Limited. 8½" x 5½", pp. 644, with 271 illustrations and four coloured plates. Price: 24s.

"Personal Mental Hygiene", by Dom Thomas Verner Moore, O.S.B., M.D., Ph.D.; 1944. New York: Grune and Stratton. 8½" x 5½", pp. 337. Price: \$4.00.

"Arterial Hypertension: Its Diagnosis and Treatment", by Irvine H. Page, M.D., and Arthur Curtis Corcoran, M.D.; 1944. Chicago: The Year Book Publishers, Incorporated. 8" x 5½", pp. 352, with 14 illustrations. Price: \$3.75, post paid.

"The 1944 Year Book of Pediatrics", edited by Isaac A. Abt, D.Sc., M.D., with the collaboration of Arthur P. Abt, B.S., M.D., Comdr., M.C., U.S.N.R.; 1944. Chicago: The Year Book Publishers. 7" x 4½", pp. 448, with many illustrations. Price: \$3.00; in Australia, 23s. 6d.

"Reagent Chemicals", by Samuel Morris, A.S.T.C. (Chem.), A.A.C.I., F.C.S.; 1944. Sydney: Grosvenor Laboratories, Pty. Ltd. 8½" x 5½", pp. 156.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmalm United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phoenix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia. All Public Health Department appointments.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot, under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such a notification is received within one month.

SUBSCRIPTION RATES—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and book-sellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rates are £2 for Australia and £2 5s. abroad per annum payable in advance.